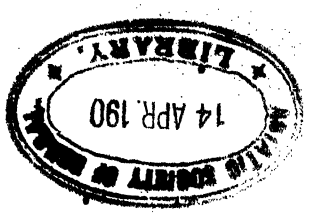
STUDIES IN CLINICAL MEDICINE.



Studies in Clinical Medicine.

A RECORD OF SOME OF THE MORE INTERESTING CASES OBSERVED, AND OF SOME OF THE REMARKS MADE, AT THE AUTHOR'S OUT-PATIENT CLINIC IN THE EDINBURGH ROYAL INFIRMARY.

BY

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Studies in Clinical Medicine.

FRIDAY, NOVEMBER 15, 1889.

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I.—HEAD INJURY; EPILEPSY; (?) GENERAL PARALYSIS OF THE INSANE.

(The patient had not been seen before he came to the Clinic.)

Dr B. What is your name?

Patient (with marked emphasis). James M———.

Dr B. How old are you?

Patient (also with emphasis). Forty-three.

Dr B. What is your occupation?

Patient (still with emphasis). A labouring man.

Dr B. Where do you live?

Patient (still with emphasis, and some hesitation and want of sharpness in articulation). 19 P——— Street.

Dr B. (*to the Students*). This patient has been sent to the Clinic by one of my medical friends, with the statement that the case is one of interest and importance; the patient, I understand, states that he received a severe head injury by a fall from the Forth Bridge some months ago, and that he has since suffered from attacks of epilepsy. I have not seen the patient until he came into the room just now, but from the manner in which he has answered the few routine questions which I have just put to him, and from certain other facts which I have observed while he was speaking, I have already formed the provisional opinion that he probably is suffering from another disease in addition to epilepsy.

You will have noticed the peculiar manner in which he has answered the questions I have put to him; he spoke with quite an unnecessary degree of emphasis and empressement; which to me, is highly suggestive of marked mental impairment or peculiarity. His articulation is also somewhat hesitating. He seems unable to pronounce some words sharply and clearly. You may, too, have noticed that while he was speaking there was every now and again a very obvious tremor or twitching of the muscles of the left side of the upper lip and adjacent portion of the face. When the face is at rest, it presents, too, a somewhat expressionless appearance; it looks flattened out, as if the facial muscles were not kept in that degree of tension which is usually present in most healthy individuals. I have noticed, too, that there is every now and again a fine tremor in the limbs, more especially in the lower extremities. Now these facts, more particularly the twitching of the facial muscles when the patient speaks, the want of sharpness in articulation, and the peculiar emphasis with which he answered such ordinary questions as his name, age, and occupation, are to my mind strongly suggestive that we have before us a case of G. P., or general paralysis of the insane. The twitching of the facial muscles, and the difficulty of articulation, which are such striking characteristics of that affection, seem well marked, and will probably be very perceptible when he repeats the following sentences: "Papa's performing pony." "West Register Street."

The patient articulated the sentences in a

characteristic manner, the tremor of the lip muscles was very apparent, and there was marked difficulty and hesitation in pronouncing the p's, the lip muscles appearing to hesitate and stumble.

Dr B. Put out your tongue.

The tongue was protruded without difficulty and without obvious tremor.

Dr B. (*to the Students*). Tremors are often noticed in the tongue as in the lips and facial muscles; but in this case they do not appear to be present.

Dr B. What do you complain of?

Patient. I take turns through the night.

Dr B. What are the turns like?

Patient. Well, I cannot say for that, because I do not know except I am awake. When I put my clothes on the next morning, I think the house is different from what it was the night before. The room seems strange.

Dr B. Does your wife say that you are convulsed in the attacks?

Patient. Yes.

Dr B. Do you yourself know when you take them?

Patient. No.

Dr B. Do you bite your tongue in the attacks?

Patient. Yes.

Dr B. Do you feel stupid and confused in the morning after an attack?

Patient. Yes; I feel quite confused and stupid in the morning, and I feel a pain in the opening of the head (placing his hand on the top of the head).

Dr B. Do you ever wet your bed?

Patient. Yes.

Dr B. Do you ever take a fit through the day?

Patient. No.

Dr B. Do you ever feel, before going to bed, anything which tells you that you are likely to have an attack through the night?

Patient. Yes, I sometimes feel a pain in my head, and a singing in the ears the night before.

Dr B. Do you ever feel that pain in your head, without having an attack through the night afterwards?

Patient. No.

Dr B. You are sure that you never suffer from headache, except before the attacks which you have during the night?

Patient. No, sir.

Dr B. (*to the Students*). That, gentlemen, is a point of importance, for it is strongly opposed to the view that the patient is suffering from any coarse lesion of the brain, such as abscess or tumour, which might have followed an injury. We will afterwards examine the optic discs, a procedure which I have so frequently told you should be practised as a matter of routine in all cases of suspected cerebral disease.

On examination, the optic discs were found to be healthy.

Dr B. Have you greater difficulty in speaking the next morning after an attack?

Patient. Yes; I often have great difficulty in speaking the next morning.

Dr B. Do you feel any weakness or numbness in your arms or legs after the attacks?

Patient. My right hand and leg often feel weak and numb the next morning.

Dr B. (*to the Students*). Temporary loss of speech or difficulty in speaking, and temporary paralysis or weakness on one side of the body, are very frequently observed after the so-called "congestive" attacks and epileptiform seizures, which are of common occurrence in general paralysis of the insane.

Dr. B. You had a fall from the Forth Bridge?

Patient. Yes.

Dr B. Were you quite well before that?

Patient. Yes.

Dr B. Was the injury a severe one?

Patient. Yes; I was brought to the Infirmary.

Dr B. Were you insensible?

Patient. Yes, for fifteen hours.

Dr B. Was your head cut?

Patient. Yes.

Dr B. Was the skull fractured?

Patient (with some hesitation). I think so.

Dr B. In which ward were you?

Patient. One of the surgical wards. I do not know which one.

Dr B. Who was the doctor?

Patient. I do not know.

Dr B. Is your memory worse since the cident?

Patient. Yes, very much; it is very bad.

Dr B. How long were you in the hospital?

Patient. Nine weeks.

Dr B. Did you take any turns through the ght while you were in hospital?

Patient. No. I began to take the turns two nths after I left the hospital.

Dr B. Have you any queer fancies?

Patient. I don't think so.

Dr B. (*to the Students*). The character of the speech, the tremor of the lips, and the facial appearance, are, as I have already stated, strongly suggestive that this patient is suffering from general paralysis of the insane. That disease, or G. P., as it is commonly termed, is *the* mental disease which has a well marked morbid anatomy. The pathological appearances are quite distinct; the convolutions are shrunken, the membranes adherent here and there to the surface of the brain, the superficial parts of the brain cortex atrophied and sclerosed. The pathological appearances in other forms of insanity are indefinite and not characteristic; indeed, we know little that is certain as to the morbid anatomy of most forms of insanity. I understand, however, that Dr Bevan Lewis, one of our greatest authorities on the histology of the brain, is about to publish a book on the morbid histology of the insane brain; and perhaps his observations may put the matter on a more satisfactory basis.

In addition to the objective symptoms, such as the characteristic alteration of speech, and the muscular weakness and tremors which I have mentioned, the mental faculties are profoundly modified, in most cases of general paralysis of the insane. The most characteristic mental alteration, though it is not always present, is a peculiar condition of mental exaltation. Everything appears to the patient *couleur de rose*. He takes an altogether too hopeful view of himself and his circumstances. He may fancy, for example, that he is some great personage, that he is enormously rich, extremely well, unusually clever or strong.

Dr B. (*to the Patient*). Have you any ideas of that kind?

Patient. I think sometimes that I am in better circumstances than I am.

Dr B. Do you ever tell people that you are in good circumstances?

Patient. I sometimes talk about it to people. I often dream that I am well off.

Dr B. (*to the Students*). That is a very interesting point. It is quite in keeping with what we know of the physiology of the brain, that a patient who has grandiose ideas, as they are termed, through the day, should have the same grandiose ideas in his dreams. In dreaming, though the patient is unconscious, or in some cases only very slightly conscious, of his surroundings, the same parts of the brain are functionating which are in activity during the process of thinking, when he is fully conscious and awake. The correct association of ideas, the fine co-ordination, so to speak, of brain action, is often, however, deranged during sleep. An idea arouses other ideas, which would either not be aroused, or if they were aroused, would, during full consciousness and brain activity, be instantly recognised as so absurd that they would be immediately suppressed. Or, to put the matter another way, during sleep the nerve discharges, which flow to, and call into action, the different and distant portions of the brain (from those in which the primary discharge is liberated), and which are associated in the production of ideas, flow in irregular and unaccustomed channels, and do not follow the beaten tracts which they are accustomed to take when the patient is awake. The physical process with which the psychical condition is associated, which we term the association of ideas, is, in fact, apt to be more or less inco-ordinate during sleep. Dreams are, however, as a rule forgotten. The ideas which are associated with the subconscious activity of the nerve cells, such as occur during the act of dreaming when the individual is asleep, do not as a rule leave a vivid and permanent impression on the nerve cells; they are not usually remembered, or capable of being called into remembrance by an act of memory, when the patient becomes awake. This is, so

far as I remember, the first case which has come under my notice in which a patient suffering from general paralysis (supposing that this patient is affected with general paralysis), has told me that he dreams grandiose ideas.

In general paralysis of the insane, the mental faculties are more or less profoundly altered. Memory, as in this case, is usually very much impaired.

It is important to remember, that grandiose ideas are not always present in cases of G. P. In some cases, the patient is melancholic. In others, there is an alternate condition of mental exaltation and melancholia. Further, it seems to be a fact, that in some patients, who present all the physical alterations characteristic of general paralysis of the insane, there are, for a time at all events, no definite and distinct mental alterations.*

Dr B. (*to the Patient*). Do you ever get depressed?

Patient. A little in that way.

Dr B. Not to any great degree?

Patient. No, sir.

Dr B. Come back on Wednesday, and bring your wife with you.

Dr B. (*to the Students*). We shall probably be able to learn from the patient's wife more definite particulars as to his mental condition.

In general paralysis of the insane, the patient may not only have grandiose ideas, but he may act on those ideas. This is a point of great importance, both for the patient and his friends. In its earlier stages, general paralysis of the insane is often unrecognised. It is one of the diseases which most frequently is unrecognised even when the symptoms are well marked. In the earlier stages, though the patient's relatives and friends may have noticed that his mental condition is distinctly changed, they may not attach any importance to the condition. Now a patient affected with general paralysis in its earliest stages, may quickly squander the earnings of a lifetime, and may involve himself and his relatives in financial difficulties. He may, in this condition of insanity, contract engagements, and involve himself in liabilities which it may be difficult or impossible to set aside. In some cases, his acts of this kind are obviously the result of insanity. He may, for example, go into a shop and give an order for fifty pianos.

In dealing with cases of suspected insanity, it is always important to ascertain if the patient has acted upon his insane ideas. An absurd or extraordinary idea may, of course, occur to any one. The sane brain sees the absurdity, and does not act upon it; but the insane brain fails to see the absurdity, reasons that the idea is not absurd, will not be convinced that it is absurd; or, prompted by the absurd idea, performs some absurd, outrageous, or altogether purposeless act, which the sane man would never allow himself to perform. The sane brain would inhibit the purposeless or outrageous act suggested by the absurd idea.

An epileptic fit is not very uncommon in the earlier stages of general paralysis of the insane; and whenever a man over thirty-five years of age, who has not been subject to epileptic fits, takes an epileptic fit, general paralysis of the insane should be thought of as one of the possible causes of the condition. A case of this kind came under my notice about eighteen months ago. A man somewhere about thirty-eight years of age, I do not remember his exact age, consulted me on account of an epileptic fit. I was unable to detect any definite disease to account for the condition, I certainly saw nothing to lead me to suspect that he was suffering from general paralysis of the insane. I saw no more of him for a year; when he again consulted me, all the symptoms, objective and mental, of general paralysis of the insane were present in a most typical degree.

Certain alterations of the pupil are very frequently present in general paralysis. The pupils may be unequal in size; the margins of the pupil may be irregular; or the Argyll Robertson condition of the pupil may be present. In this particular case (Dr Bramwell here examined the pupils), the right pupil is somewhat smaller than the left, but its margins appear to be even (regular), and the

* I have at present, under observation, a very remarkable case of this kind, which will be referred to in a subsequent number of these *Studies*.

reflex contractility to light is active. In the Argyll Robertson condition, the pupil, as you know, fails to contract to the stimulus of light, while it still contracts when the eyeballs are rotated inwards during an act of accommodation for near vision. Dr Argyll Robertson first described this condition of the pupil in cases of locomotor ataxia. It is a very valuable sign of locomotor ataxia, for it is present in a considerable proportion of cases, and it usually occurs in the earlier stages of the disease, when the diagnosis is most obscure. The same condition of the pupil is frequently present in general paralysis of the insane. In fact, when you meet with the Argyll Robertson condition of the pupil, you should always suspect either locomotor ataxia or general paralysis of the insane; for although it doubtless may occur in other affections, it is very much more common in these diseases than in any other conditions.

General paralysis of the insane is a most incurable affection; indeed, many authorities deny that it is ever recovered from. Most cases die within two or three years. In some cases, remissions which may be mistaken for recoveries are met with; during the remission, the mental condition and other symptoms may become so markedly better, that the remission or temporary condition of improvement may be thought to be a permanent cure. I have recorded one very remarkable case in which the symptoms were typically characteristic of general paralysis of the insane, and in which the patient has now for more than six years been practically well. Since his recovery he has been able to follow his employment,—that of an engineer's draughtsman. The only symptom indicative of the former brain mischief, which now remains, is the occasional occurrence of an attack of *petit mal*.

In the case to which I refer, the patient had had syphilis, and there can, I think, be little doubt that the brain disease was syphilitic. But be that as it may, the symptoms were characteristic of general paralysis of the insane. My diagnosis of G. P. was corroborated by Dr Tennant, of Glasgow, and Dr Clouston. In his admirable book on Insanity, Dr Clouston refers to the case as a typical example of general paralysis of the insane. The case is one of great importance, for it shows that a condition which cannot be distinguished clinically, during life, from general paralysis of the insane may be the result of syphilis, and may be recovered from under a vigorous anti-syphilitic treatment.

The details of the recovery in that case are interesting; the patient, notwithstanding the free administration of iodide of potassium and mercury, for a long time, seemed to make little or no progress. He had during this period several of the so called congestive attacks, in which he was for a time unconscious, and which were followed by temporary hemiplegia or aphasia. The last of these pseudo-apoplectic attacks was an unusually severe one. He remained unconscious for many hours—I think thirty-six. A large acute bed sore formed on the sacrum and buttocks; this was followed by extensive suppuration and sloughing; the separation of the slough was attended with profuse hæmorrhage, which was arrested with difficulty. After emerging from this very grave condition, he progressed steadily and uninterruptedly towards recovery, and in the course of a few months was sufficiently well to return to business.

It is important to note that, by Dr Tennant's advice, he steadily continued to take the iodide and mercury after his return to business; indeed, when I last heard of him, now more than a year ago,—some five years after his recovery and return to work,—he was still taking the iodide at intervals.

The exact influence which syphilis has in the production of general paralysis of the insane is a disputed point. Some authorities think that syphilis is, in many cases, an important factor in the production of general paralysis. Others, and Dr Clouston is one of the strongest supporters of this view, think that it has little or no influence in the production of *the* disease general paralysis of the insane; though they admit that a combination of clinical symptoms, which it is impossible to distinguish from general paralysis, may be produced by

syphilis. For my own part, I am disposed to think that syphilis is, in some cases of general paralysis, an etiological factor of importance, just as it undoubtedly is in many cases of locomotor ataxia. I do not say that syphilis plays such an important part in the production of general paralysis as it does in the production of locomotor ataxia, but in a certain proportion of cases, I believe it is an etiological factor of importance. The influence of syphilis in the production of both of these diseases is probably rather an indirect than a direct one. In neither case, is the lesion definitely syphilitic and curable by anti-syphilitic remedies, as a distinctly syphilitic lesion, such as a gumma, is.

General paralysis of the insane and locomotor ataxia are rare in women, and it is very important to note that when these affections do occur in women, there is often a distinct history of syphilis, or the patient has led a dissolute and irregular life, and is therefore likely to have been exposed to syphilis. This has always appeared to me a strong argument in favour of syphilis being a cause, though it may be an indirect cause, of general paralysis. General paralysis of the insane is, in my experience, more common in the north of England, more especially in the county of Durham, than it is here. I remember meeting with at least four cases in women, while in practice in the north of England, but I have only seen one well marked case in a woman since coming to Edinburgh. All the four north of England cases occured in women who had either had syphilis, or who had led dissolute lives.

General paralysis of the insane and locomotor ataxia are not unfrequently combined. In some cases, the ataxia symptoms precede the cerebral symptoms; in others, the locomotor ataxia is developed after the general paralysis. The condition of the knee jerk is of importance in general paralysis of the insane. In some cases of G. P., in which there are no other symptoms or signs suggestive of locomotor ataxia, the knee jerk is abolished. In other cases of G. P., the knee jerk is increased.

In this patient the knee jerk was markedly exaggerated.

Alcoholic and sexual excesses have also been thought to be causes of general paralysis. I have been told that general paralysis of the insane is almost, if not entirely, unknown in persons who have lived all their lives in the glens of the Highlands of Scotland; but that the sons of the Scotch Highlanders, who go south, into the army or into business, not unfrequently return with general paralysis and die from it. I have also been told that, in Belfast, general paralysis is almost, if not entirely, unknown. Now sexual excess, alcoholic excess, and syphilis, are by no means unknown either in the Highlands of Scotland or in Belfast.* It is obvious, therefore, that if the statements which I have just made, on what I believe to be reliable authority, as to the rarity of G. P. in the Highlands of Scotland and in Belfast, are facts, general paralysis of the insane must be due to something more than mere sexual excess, alcohol, or syphilis.

One of the most important causes of general paralysis of the insane seems to be brain strain.

Unlike many forms of insanity, general paralysis of the insane is rarely directly hereditary. It is very rare to meet with two cases of general paralysis in near members of the same family. Curiously enough, I had a letter from Dr Clouston, only a few days ago, about a case of this description. Some six months ago, I saw, with Dr Elder of Leith, who was at that time taking charge of Dr Struthers' practice, a case which I diagnosed as general paralysis of the insane, and learned that the patient's brother (who like my patient was a steam-ship captain, and a very able, active, energetic, and pushing man), had been sent, some few years previously, to Morningside, with symptoms which seemed

* Since this paragraph was written, I have had the advantage of learning Dr Clouston's experience on this point. Dr Clouston tells me that G. P. is undoubtedly very rare in the Highlands of Scotland,—in persons who live in, and have never been out of, the Highlands; and that Highlanders who go south, not uncommonly become affected with G. P. He further states, that the disease is almost unknown in the west of Ireland, but that it does certainly occur in Belfast. At the present time, he has under his care two patients from Belfast, who are affected with general paralysis of the insane.

to shew that he also was affected with the same disease. About a week ago, I had a letter from Dr Elder, telling me that our patient had, after my visit, steadily become worse, and had been sent to Morningside. I accordingly wrote to Dr Clouston, asking him about both patients. He tells me, that the brother died in the asylum of general paralysis of the insane, and that my patient is a notable general paralytic. In his letter, Dr Clouston refers me to a paper by himself and Dr Savage, in the April number of the *Journal of Insanity*, in which the occurrence of general paralysis of the insane *in twins* is for the first time recorded.

The same Patient, March 9th, 1889.

Dr B. (*to the Students*). You remember, gentlemen, that I entered into this case with some detail a week ago; and stated, that the case seemed to be a case of general paralysis of the insane. After the Clinic was over, I learned from the nurse, what I did not know before, that the patient has been on several occasions an inmate of the hospital.

He was, so far as the nurse has been able to ascertain, first admitted in June 1886, suffering from hysteria.

In November 1886, he was again admitted, suffering from hystero-epilepsy. He remained on this occasion three weeks under treatment.

In March 1888, he was a third time admitted, for symptoms which were thought to be due partly to hysteria and partly to alcohol.

Now, you will remember that the patient distinctly stated, that he was quite well until he fell from the Forth Bridge eleven months ago, and that his epileptic fits had only developed some months after this injury.

Obviously, therefore, the patient's statements as to the history of his injury are not to be relied upon. In consequence of the uncertainty as to the facts of the case, I necessarily felt much more hesitation than I had done, when I examined the case before you, as to the nature of his illness; and was more than ever desirous of seeing his wife, and of obtaining from her exact information as to the character of his mental condition, and the duration and course of his illness.

Unfortunately, the patient did not bring his wife here on Wednesday, so that I have not been able to question her in your presence; but I called at his house yesterday, saw his wife, and elicited a number of very important and interesting facts regarding his condition.

She tells me that, until eight or nine years ago, he was a very active and smart man, and always steady. He has never, either before or since his illness commenced, indulged in any alcoholic excess; latterly, he has been in such poor circumstances, that he has had no money to spend on drink, even if he had wanted to do so.

His relations are, his wife states, fine, healthy people. His wife's mother, who lives with him, stated that his relatives are superior people. She used the words "genteel people, in good circumstances." None of his near relatives, father, mother, brothers, or sisters, have suffered from nervous disease. His mother is still alive, and is a very nice old woman. (I use, as far as possible, the exact words, which I noted down at my interview with his wife and her mother.)

The patient himself has never had any great mental strain, worry, or anxiety. Until his illness commenced, he was always in good work, earning good wages; he has always been a proud man, and very particular as regards his dress.

Some eight or nine years ago he fell down a stair and received a severe head injury. He was unconscious for many hours, and was said, by the three doctors who attended him, to be suffering from concussion of the brain.

His wife thinks that this injury was the cause of his present illness. Since the fall, he has never been exactly the same as he was before; but there was no very marked alteration in his general health or mental condition until four or five years ago, when he began to take the epileptic fits, from which he still suffers.

He usually takes the fits through the night. He has only once taken a fit in the street; but, on several occasions, he had a fit in the rubber-works, where he was employed. He

often bites his tongue severely in the fits. He very often does not seem to know when he has had a fit; this is more especially the case when the fits come on, as they usually do, through the night. The next morning after a fit he seems stupid and confused, and often complains of a pain on the top of his head. His wife states that before a fit he sometimes seems to feel cold, and shivers, even if he is sitting before a good fire.

After a fit he will sometimes get up, go out, and wander about, without knowing what he is doing.

He has been two or three times admitted into the Infirmary in an excited condition after a fit. It was thought that he had been drinking, but this was not the case.

About eleven months ago he fell at the Forth Bridge, where he was working. He hurt his shoulder, but only slightly; he did not hurt his head. He was in the Infirmary for some time after this fall.

Since the fits commenced, but more especially during the last few months, his mental condition has become much changed. Of late, the mental change has been so marked, that comparative strangers see a great change in him. His wife and his brothers and sisters do not think he is right in the mind. His wife's exact words were, "I don't think his mind is right." He has almost entirely lost his memory. His wife says, I don't think he remembers the ages of his children, and I don't think he could tell his own age if you asked him. He has ceased to take any notice of his wife and children. He never reads now, though he used to be a great reader. His wife stated that "he used to be an awful man for books and newspapers." He passes people, whom he used to know quite well, in the street without noticing them, his mind apparently being occupied, his wife says, with something else. His old friends have asked her if he is not "queer."

His mental condition is very variable. Sometimes he is depressed, and will sit for hours or even days in a chair, without taking notice of anything or anybody.

"At other times he gets quite high;—he is either awfully high or awfully low" (these are the wife's exact words). He sometimes gets much excited, and speaks very high and loud about what he is going to do. He often talks in a hopeful way of what he will do when he gets employment, and when he makes money. He says "that he will go to a better house than this hole," in which he lives; that he will be well dressed. "He sometimes imagines that he will do great things." Yet, for some time, he has done nothing, and is obviously getting worse.

He is still very paticular (his wife evidently thought absurdly particular) about his dress; for she states, he will pay great attention to his collar and neck tie, when his clothes are wretched and he has hardly a shoe to put upon his feet.

He has become very weak; yet, on the 1st of February (1889), he walked all the way to Dundee, where his sister lives, in a terrible storm. His wife does not know how he managed it, or how long he was on the road.

Asked if she has noticed any difference in his speech, or any trembling about his face, his wife promptly replied, "Oh yes, there is a catch in his speech; and his lips tremble, especially when he gets excited. He is at times awfully shakey, just like a man coming out of delirium tremens."

"He sleeps well, and eats well; but of late he has not had enough to eat."

You see, gentlemen, from this statement, that the patient has suffered from epileptic fits for the past four or five years; and the question arises, whether the mental deterioration is merely the result of epilepsy, or whether there is not general paralysis of the insane in addition. Loss of memory is a very common result of epilepsy; and after long continued epilepsy, all the mental faculties may become affected, and a condition of dementia be produced. But, so far as I can judge, there is more than epilepsy in this case. The alteration in speech, the twitching of the facial and lip muscles, when the patient speaks, and the condition of mental exaltation, which seems, from his wife's state-

ment, to be every now and again present (and not only present immediately after a fit), lead me to think that epilepsy is not the whole explanation of this case, but that the patient is also suffering from general paralysis of the insane. I do not, however, feel able to speak so certainly as to the presence of general paralysis, as I did at the time of the patient's first visit. At that time, I was under the belief that the symptoms had all developed during the past few months—since the fall and injury to the head, which he stated he had received eleven months ago. You will remember, that he stated that he was quite well before his fall at the Forth Bridge. It now appears that he has for the past four or five years been subject to epilepsy; and that his brain disease has perhaps resulted from the injury which he received eight or nine years ago.

II.—Stricture of the Œsophagus, the result of swallowing Caustic Soda; Diseased Conditions resembling Acute Irritant Poisoning.

Man; aged 31; worker in a jute mill; October 16, 1889.

Dr B. What do you complain of?

Patient. I drank some caustic soda in mistake for water; and I have pain and difficulty in swallowing.

Dr B. When did you swallow the caustic soda?

Patient. On the 26th of August.

Dr B. How much caustic soda did you swallow?

Patient. I took a mouthful.

Dr B. Did you feel any pain as it was going down?

Patient. Yes; a burning pain.

Dr B. Had you any pain afterwards in the stomach?

Patient. Yes, still a burning pain.

Dr B. Did you vomit?

Patient. I got medicine to make me vomit before I went to the Infirmary. I was taken to the Infirmary at Dundee. I was in the Infirmary seven weeks.

Dr B. Did you vomit any blood?

Patient. Yes.

Dr B. How much?

Patient. About a pint.

Dr B. Was the blood bright or dark?

Patient. Dark.

Dr B. Like coffee grounds?

Patient. Yes.

Dr B. Did you pass any blood by stool?

Patient. No.

Dr B. Were you feverish afterwards?

Patient. I began to tremble when I was put to bed.

Dr B. Did you feel cold and shiver?

Patient. Yes.

Dr B. Did you continue to suffer from your stomach for some time?

Patient. Yes.

Dr B. Had you any difficulty of swallowing when you left the Infirmary?

Patient. No, it came on afterwards; I went back to the Infirmary.

Dr B. Have you any pain when you swallow now?

Patient. Not if I confine myself to taking corn flour, everything else hurts me.

Dr B. Where does it hurt? where does the food seem to stick?

Patient (pointing to the upper part of the sternum and lower part of the neck). There.

Dr B. Have you lost much flesh?

Patient. Yes; I am very weak.

Dr B. (*to the Students*). The case is one of great interest. Cases of this kind are comparatively rare. There can, of course, be no doubt that the patient is suffering from the injury to the œsophagus produced by the caustic soda. There is evidently a stricture, and a painful stricture. The question comes to be, whether the obstruction is simply the result of cicatricial contraction of the injured walls of the œsophagus, or whether the narrowing of the gullet is due, in some measure at least, to the presence of an abscess or of inflammatory

products, in the wall of the œsophagus or in the connective tissue which surrounds it. The fact that there is pain, as well as difficulty in swallowing, seems to shew that there is more than a simple cicatricial stricture. There is evidently some inflammatory condition at the seat of the stricture. It is impossible, I think, to say whether that inflammatory condition is confined to the œsophagus. There may be an abscess in the wall of the œsophagus, or an abscess outside it, which is pressing upon and narrowing the gullet.

A simple stricture is by far the rarest form of organic stricture of the œsophagus. Difficulty of swallowing, the result of obstruction in the gullet, may be due to a variety of different causes. Organic strictures are one cause. The pressure of aneurisms, solid tumours, inflammatory collections on the outside of the œsophagus, is another. Spasmodic strictures sometimes occur, more especially in hysterical subjects.

In most cases, in which the obstruction is organic, and situated in the wall of the œsophagus itself, the stricture is due to malignant disease. When the stricture is simple, there almost always is a history of injury to the wall of the gullet. The swallowing of corrosive acids or alkalis, or of boiling water, is the usual cause of such injury. Children not unfrequently scald themselves by swallowing boiling water (taking a suck out of the kettle spout, for instance). In some cases, the œsophagus is scalded, and a simple stricture results—just as a simple stricture of the urethra results after gonorrheal inflammation.

The condition from which this patient is suffering will require careful watching and treatment.

Dr B. Do you wish to get into the hospital?

Patient. Yes.

Dr B. Did any of the caustic soda run on to and stain your clothes?

Patient. No.

Dr B. Did it hurt your lips, mouth, and throat?

Patient. Yes, the mouth and throat were very much inflamed and swollen.

(The patient was then sent up to the wards.)*

Dr B. (to the Students). When a patient complains of sudden pain in the stomach and vomiting, and becomes collapsed, one should always suspect that the case may possibly be one of acute irritant poisoning. Under such circumstances, the first thing which we have to ascertain is if the pain and vomiting came on after eating or drinking anything. In a case such as this, in which a corrosive acid or alkali has been

* After seeing this patient I wrote to Dr Erskine, the House Surgeon to the Dundee Infirmary, asking him if he could give me any information about this patient, and telling him that I intended to refer to the case in these *Studies*. Dr Erskine has very kindly sent me the following notes of the case:—

"The patient was admitted at 5 P.M. on August 26th. He stated that while engaged at his work in South Dudhope Jute works, he had, just before being brought to the Infirmary, drank one mouthful of a solution of caustic soda, which was standing near an oil tank, for the purpose of making a soap to put on the jute.

"He complained of pain all down the gullet, most severe in the region of the stomach.

"The tongue and pharynx were bright red in colour, and denuded of their epithelium. Deglutition was painful and difficult. The patient was somewhat collapsed; the pulse was 60, regular, but weak.

"He was made to swallow some vinegar and water, and some olive oil, and a poultice was applied to the epigastrium. Milk was ordered as food. The evening temperature was 98° F.

"*Aug.* 27—Passed a good night; slept well; expectorating muco-pus; M.T., 99.2°; ice to suck; to gargle the throat with warm water; E.T., 100°.

"*Aug.* 28.—Swallows very well; the pain during deglutition is confined to the fauces; taking a good quantity of milk and beat-up eggs; moderate expectoration of muco-pus; M.T., 99.2°; E.T., 100.2°.

"*Aug.* 29.—M.T., 99.2°; E.T., 99 4°.

"*Aug.* 30.—Still some purulent expectoration; to have beef tea; M.T., 98.8°; E.T., 99.4°.

"*Aug.* 31.—M.T., 98.4;° E.T., 99°.

"*Sept.* 1.—M.T., 98.8°; E.T., 101.6°.

"*Sept.* 2.—M.T., 99.2°; E.T., 99.4°.

"*Sept.* 3.—Raw surface skinning over; general condition favourable; M.T., 98.4°; E.T., 98.8°.

"*Sept.* 4.—Can get up; M.T., 98.4°; E.T., 98.4°.

"*Sept.* 7.—Can go home. Advised to come back if any difficulty of swallowing should come on.

"*Sept.* 11.—Re-admitted, complaining of being unable to swallow any solid food. Even very minute pieces of solid food stick in his throat, and cause great pain.

"*Sept.* 20.—Can only swallow milk.

"*Sept.* 21.—A narrow œsophageal bougie was passed down the gullet for 14 inches. This caused great pain, and the patient went home in the afternoon by desire."

swallowed, the pain and vomiting come on immediately after the act of swallowing, and the passage of the corrosive liquid down the gullet is usually attended with great pain and burning. The lips, tongue, mouth, and throat, are usually more or less excoriated; and the effects of the acid or alkali may often be seen on the clothes. It is very important in cases of suspected acute irritant poisoning to examine carefully the clothes, the lips, and the interior of the mouth and throat. The fact that the clothes are burned or stained, and that the lips, mouth, or throat are excoriated, determines the diagnosis. In the case of some other irritants, arsenic for example, the pain and vomiting may not come on immediately after the poison is swallowed, and there are no stains on the clothes, and no excoriations on the mouth to guide one. The patient does not usually complain of any pain in the gullet—the act of swallowing the poison may of course be absolutely painless.

The fact that the pain in the stomach and vomiting (in a case in which a patient who is suddenly seized with pain in the stomach and vomiting, and who becomes collapsed) occurred immediately after, or soon after, taking food, drink, or medicine, is suggestive that the food contained some irritant, possibly an irritant poison. It is not, however, conclusive. There are many diseased conditions which resemble more or less closely,—and sometimes very closely—acute irritant poisoning. When we are called to such a case as I am describing, it is of very great importance to have at one's finger ends the various diseased conditions which may possibly be the cause of the symptoms. Pathological and clinical knowledge of this kind is invaluable for diagnosis. If you know all the different possible conditions, which may produce such a set of symptoms, the diagnosis is rendered very much easier than it otherwise would be. You very soon come to see some symptoms or signs which enable you to narrow the inquiry. You may in this way be able to exclude first one thing and then another, or to see something which decides the diagnosis in favour of one cause or another

Now in a suspected case of acute irritant poisoning—when a patient is suddenly seized with pain in the abdomen, and especially in the region of the stomach, when there is vomiting and collapse,—the first question which we have to try and decide is, whether the symptoms are the result of acute irritant poisoning or of disease. I do not propose to enter into details as to the differential diagnosis of the different forms of acute irritant poisoning. That information you get in your lectures on medical jurisprudence; but the differential diagnosis of irritant poisoning and diseased conditions simulating it, is a matter which belongs to medicine quite as much as to medical jurisprudence. It is a very interesting subject. When I held the chair of medical jurisprudence in the University of Durham, I used to go into great detail regarding this point. The text books on medical jurisprudence do not, so far as I am acquainted with them, consider the subject so fully as, in my opinion, it deserves to be considered. I have personally met with a variety of different cases in which this question of diagnosis has had to be determined, either during life or after death.

The first point, as I have already said, is to ascertain whether the symptoms can be directly attributed to anything which has been taken in the form of food, drink, medicine, &c. The fact that the pain in the stomach and vomiting did occur immediately after, or soon after, eating or drinking, is suggestive of acute irritant poisoning, but by no means conclusive. I will presently mention to you some cases illustrative of this point.

The character of the vomited matters is an important point. The fact that the vomited matters contain blood—have the coffee-ground character—granting that we can exclude ulcer of the stomach (one of the diseased conditions which most closely resembles irritant poisoning), is very strongly in favour of irritant poisoning. It is important, however, to remember, that coffee-ground vomiting, or the vomiting of blood, is by no means always present in acute irritant poisoning.

When there is no blood in the vomit, the

character of the vomited matters usually fails to give any definite information, until a chemical analysis is made. Now, few practitioners are able to make a satisfactory chemical analysis; and the few, who have the knowledge, do not, of course, carry chemical reagents and apparatus about with them in private practice; so that, for the purposes of immediate diagnosis, the chemical analysis may usually be left out of account.

It is very important, however, to remember, that in every case of suspected irritant poisoning, the vomited matter should be carefully collected, sealed up, and set aside for future examination. For the same reason it is of the utmost importance, when it can be obtained, to secure some of the food or drink which the patient took, just before he was seized with the symptoms. It is important also to secure, for chemical analysis, any urine which is passed after the symptoms develop. It is impossible to overrate the importance of being alive to these points.

The occurrence of diarrhœa, as well as vomiting, is suggestive of irritant poisoning, but by no means conclusive, as we shall presently see.

The fact that *several* persons have been simultaneously attacked with symptoms suggestive of irritant poisoning (pains, vomiting, &c.), soon or immediately after partaking of a meal, is, of course, most important corroborative evidence in favour of poisoning. It is not, however, conclusive. I will presently mention a case in point.

The fact that the symptoms occurred suddenly in the midst of apparent health, is highly suggestive of acute irritant poisoning. It is not, however, conclusive. Some diseased conditions, which produce symptoms closely resembling those of irritant poisoning, develop suddenly, or are latent at all events until the final outburst, so to speak, of acute symptoms occurs.

A careful inquiry into the previous state of health of the patient is of the utmost importance, in many cases, for the purposes of differential diagnosis. In the great majority of cases of disease, in which symptoms suggestive of irritant poisoning suddenly develop, careful inquiry into the previous history will shew that the patient, before the appearance of the symptoms, was obviously out of health. It will be ascertained, in most cases, at all events, that he was suffering from some symptoms suggestive of those diseased conditions, in the course of which complications or accidents, likely to be attended with the sudden occurrence of pain in the abdomen, vomiting, and collapse (the symptoms suggestive of irritant poisoning), are apt to arise.

The fact that the patient has been suffering from such diseased conditions does not, of course, absolutely exclude acute irritant poisoning, for a diseased person may be poisoned as well as a healthy person; but it renders the probability of poisoning unlikely. It is very strongly in favour of disease rather than of poisoning.

Let me now refer to some of the diseased conditions in which symptoms suggestive of acute irritant poisoning may occur. I shall only allude to those conditions in which symptoms suggestive of irritant poisoning may occur in the midst of what appears to be good or perfect health. I shall leave out of account all those conditions in which the patient has obviously been ill before the symptoms suggestive of poisoning developed—such conditions, for example, as typhoid fever or tubercular ulceration of the intestine. In typhoid ulceration, more especially, a sudden perforation of the gut, may be attended with acute pain in the abdomen, vomiting, and collapse. But the fact that the patient was known to be suffering from typhoid—in the course of which perforation with the symptoms I have mentioned is of not infrequent occurrence—would at once negative any idea of irritant poisoning. No man of ordinary common sense and good judgment would think of irritant poisoning under such conditions, unless there were some suspicious circumstances suggestive of that condition. Irritant poisoning might, of course, occur in the course of typhoid or other diseased conditions, in which vomiting, pain in the abdomen, and collapse may arise suddenly as the result of natural causes, *i.e.*, of disease. The

differential diagnosis of such cases—fortunately very rare indeed*—might be impossible. Irritant poisoning under such circumstances would probably escape notice; the symptoms would naturally be thought to be the result of the disease. It would only be a very sharp and acute observer, thoroughly skilled and acquainted both with the symptoms of disease and with the symptoms of irritant poisoning, who would under such circumstances be likely to suspect or diagnose poisoning.

Violent vomiting, diarrhœa, collapse, and in some cases pain in the stomach or abdomen, are sometimes due to eating poisonous fungi, putrid meat, stale fish, &c. In such cases the symptoms may very closely resemble those of poisoning by arsenic or other metallic irritants.

Milk which is in a state of decomposition may produce in children the most violent vomiting and diarrhœa. I remember one of my own children being suddenly seized with alarming symptoms of this kind after taking a drink of milk. I satisfied myself that the milk was in a state of decomposition, and that it did not contain any ordinary inorganic poison. The differential diagnosis of cases of this kind and of ordinary irritant poisoning, such as arsenical poisoning, is in some cases impossible without a chemical analysis. In poisoning by a metallic irritant, pain in the stomach is, I think, usually greater and perhaps more localised than in those cases in which the symptoms result from poisoning by fungi, putrid meat, &c. Nervous symptoms, too, are much more apt to be produced, where the irritant is a ptomaine, or poisonous fungus. In some cases indeed of poisoning by fungi, the symptoms rather resemble those of a narcotic than an irritant poison.

The differential diagnosis of chronic irritant poisoning (poisoning by arsenic, corrosive sublimate, &c.), in a diseased person is also a subject of great difficulty, but of real importance, as the celebrated Maybrick case has so recently shewn. In some future number of these *Studies* I will consider the differential diagnosis of chronic irritant poisoning and of disease,—a subject which was referred to but not considered in detail at this Clinic.

The following, then, are some of the more important diseased conditions, which may be altogether, or apparently altogether, latent, and in the course of which symptoms (such as vomiting, pain in the abdomen, collapse, and possibly diarrhœa), suggestive of acute irritant poisoning may arise.

Simple or perforating ulcer of the stomach.—Several cases, in which the symptoms attending the sudden perforation of a simple ulcer of the stomach, have been thought to be due to acute irritant poisoning, have come under my own observation. I will mention one or two.

Several years ago, I was asked by my friend Mr Page, at that time police-surgeon in Newcastle, to assist him in making a *post-mortem* examination on the body of a servant girl, who had died with symptoms which it was thought might have been the result of poisoning. She had, it was stated, been perfectly well until the day before. So far as we could ascertain, she had, prior to her last sudden illness, made no complaint. So far as was known, she had not suffered from any of the symptoms of gastric ulcer. The day before her death she was seized, soon after eating her dinner, with violent pain in the region of the stomach; she vomited repeatedly; became collapsed, and went to bed. Some warm fomentations were applied over the abdomen, and some stimulants and laudanum, if I remember right, were given to her. She was not seen by any medical man. How she passed the night was not known, but the next morning she was found dead in bed. The death, you will observe, was unusually rapid. The Coroner was very naturally communicated with; and Mr Page and myself were asked to investigate the case.

We found the patient a remarkably well-nourished young woman. On opening the abdomen, diffuse peritonitis was found to be present; the contents of the stomach were extravasated into the cavity of the peritoneum; and the cause of the extravasation was found to be a perforating ulcer of the stomach. I have

* I am referring to acute rather than chronic irritant poisoning.

the specimen in my museum (see figs. 80 and 81). It is as typical and beautiful an example of simple perforating ulcer of the stomach as it is possible to see. The peritoneal surface of the ulcer is remarkably clean cut and well defined; it presents the characteristic gouged-out appearance, described in the books, in a very typical way. The gastric surface is also quite characteristic; the great size of the ulcer at the level of the mucous surface of the stomach, and the gradual diminution by a series of stages or steps, from the surface of the stomach, through the muscular coat, to the peritoneal surface, is admirably shewn.

Another very striking case of the same kind (a simple ulcer of the stomach, suddenly perforating, the ulcer being so far as was known unattended with previous symptoms) occurred when I was pathologist to this Infirmary. A big robust policeman, one of the biggest and finest men in the force, who so far as I remember, from the account which Dr Affleck gave to the students at the autopsy, had not previously complained of being ill, was brought into the Infirmary, suffering from peritonitis. He died, and on making a *post-mortem* examination I found exactly the same state of matters. Dr Affleck, in whose ward the patient was, has, I believe, the specimen in his possession.

After the publication of the first number of these *Studies*, a medical friend kindly sent me a cutting from a newspaper, in which a case of supposed irritant poisoning is recorded, the symptoms being also due to the sudden rupture of a gastric ulcer.

The report of the case, as reported, is as as follows:—

"*The alleged suspicious death of a servant girl.*—The Borough Coroner (Dr D. Wightman) held an inquest at the New Inn, Ecclesall Road, yesterday, as to the cause of death of Mary Wilson, twenty-three, who was a domestic servant in the employ of Mrs Margaret Smith, widow, Broomhall Place, and who died, under circumstances that were thought to be suspicious, on Wednesday morning last.—Isaac Wilson, wheelwright, said he had resided about thirty-five years at Dore, but he had just gone to live at Fulwood. His daughter had been fairly healthy until the last few weeks, when she had complained of pains in her inside. Eleven weeks ago, she visited her home, and he had not seen her since.—Mr Lockwood, surgeon, Abbeydale Road, said he had made a post-mortem examination of the deceased. The cause of death was peritonitis and shock, caused by a ruptured ulcer of the stomach. The ulcer might have been there for some length of time, and would produce great pain. There was no suspicious circumstance about her death.—The Coroner said he thought he need call no further evidence. The girl, when taken ill, was cleaning the fire grate, and as soon as Mrs Smith thought she was seriously ill, she sent for Dr Thomas. That gentleman prescribed for her, but she died after having been seen by the doctor's assistant. The evidence of Mr Lockwood was conclusive. The jury returned a verdict accordingly."

In the policeman's case there was not, so far as I know, any suspicion of poisoning. I mention it simply to show you that an ulcer of the stomach, which has apparently been latent, may suddenly rupture, and produce violent symptoms.

Now in the great majority of cases in which an ulcer of the stomach ruptures, the previous history of the case—a history of dyspeptic symptoms, vomiting, and pain after eating, vomiting of coffee-grounds, anæmia, etc.—gives very important information, which enables us to diagnose or suspect ulcer, and so to exclude acute irritant poisoning. But in some cases, there are no previous symptoms; or, what is perhaps more likely, in most cases of this kind at all events, the patient has not been known to complain of symptoms suggestive of ulcer of the stomach.

If the patient were a servant girl, ulcer and perforation should be suspected; for, as we all know, simple perforating ulcer is very common in such persons. The mere age and occupation of the patient are only, however, minor points. A servant girl may of course be poisoned as well as any one else.

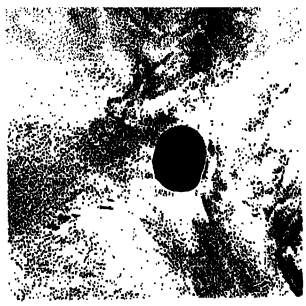


FIG. 80.—Perforating ulcer of the stomach, showing the clean-cut, punched-out appearance of the opening in the stomach, as seen from the peritoneal surface.

FIG. 81.—Perforating ulcer of the stomach, seen from the interior of the organ. A probe projects into the stomach from the pyloric orifice. The large superficial extent of the ulcer, at the level of the mucous membrane, and the gradual way in which it shelves down and narrows in extent, until it reaches the peritoneal coat, are admirably shown.

Studies in Clinical Medicine.

FRIDAY, NOVEMBER 29, 1889.

CONTENTS.

I.—THE DIFFERENTIAL DIAGNOSIS OF ACUTE IRRITANT POISONING, AND OF DISEASED CONDITIONS RESEMBLING IT.

(*Continued from page* 200.)

Simple perforating ulcer of the duodenum is a much rarer condition than simple perforating ulcer of the stomach.

The sudden rupture of an ulcer of the duodenum into the cavity of the peritoneum is perhaps even more likely to be mistaken for acute irritant poisoning, than the sudden rupture of an ulcer of the stomach; for a simple perforating ulcer of the duodenum is more frequently latent,—unattended with definite and distinct symptoms,—than a simple perforating ulcer of the stomach.

Simple perforating ulcer of the duodenum, unlike simple perforating ulcer of the stomach, is more common in men than in women, and more common at or after middle age than in young adult life. The symptoms may be very indefinite. Pain or uneasiness is in most cases complained of, but as a rule it comes on at a considerable interval, two hours or more, after taking food; while in simple perforating ulcer of the stomach, the pain or uneasiness is usually complained of immediately or soon after eating. In some cases of perforating ulcer of the duodenum, little or no pain is complained of. Vomiting may be entirely absent. In many cases, however, there is the sudden discharge of a large quantity of blood into the duodenum. In some cases, the blood regurgitates into the stomach, and is expelled by vomiting; in others —and this is most frequent—there is no vomiting of blood, but some hours after the occurrence of symptoms indicative of internal bleeding, a large quantity blood of is passed by stool. Usually, palpation will elicit the presence of some tenderness on pressure in the situation of the duodenum. In some cases, in which the ulcer is situated in close proximity to the orifice of the common duct, there is jaundice. This symptom is usually the result of the cicatricial contraction of the ulcer, narrowing or obstructing the orifice of the duct, and so preventing the passage of bile into the intestines.

I have been induced to describe the chief symptoms and signs of simple perforating ulcer in detail, because the condition is rare, its clinical features are in many cases very obscure, and the subject is one with which many practitioners are very imperfectly acquainted.

The diagnosis of perforating ulcer of the duodenum is in many cases impossible during life. The condition may be suspected, but cannot be positively diagnosed, unless the ulcer has opened into a vessel, and given rise to hæmorrhage. The occurrence of symptoms and signs indicative of internal hæmorrhage, followed by the passage of tarry stools, is the only symptom of real diagnostic value. It must be remembered that, when a large vessel is opened into, some of the blood may regurgitate into the stomach, and may be expelled by vomiting, as well as by stoc

The differential diagnosis of perforating ulcer of the stomach and of perforating ulcer of the duodenum, may under such circumstances be impossible. The facts that the patient is a male, middle aged, and that the pain or uneasiness in the region of the stomach occurred, not immediately, but at an interval, after taking food, should suggest the possibility of the ulcer being duodenal. But a positive diagnosis cannot be given on these grounds, for the same conditions *may* be present when the ulcer involves the stomach.

If these symptoms occurred in a middle-aged man, who had previously complained of obscure dyspeptic symptoms (pain or uneasiness in the region of the stomach or duodenum, the pain coming on, not immediately after, but at an interval after, taking food); if the patient were well nourished, and did not present the emaciated and cachectic appearance which is usually associated with malignant disease; and if there was no tumour or cancerous growth to be detected in the abdomen, a positive diagnosis of simple perforating ulcer of the duodenum might probably be ventured upon.

In one case, which presented these features, and in which I definitely diagnosed the presence of a simple perforating ulcer of the duodenum, the correctness of the opinion was verified by post-mortem examination. In another case, in which I also gave a positive opinion, the patient died, with symptoms of profuse hæmorrhage—but a post-mortem examination was not obtained.

Exceptionally the rupture of an ulcer of the duodenum may be attended with immediate (sudden) death. I know of one case in which sudden death was produced in this way; the patient was a medical man; and curiously enough, in another case of perforating ulcer of the duodenum, which came under my observation some ten years ago, the patient was also a medical practitioner.

Now, the sudden rupture of a simple perforating ulcer of the duodenum into the cavity of the peritoneum may, as I have already stated, be attended with symptoms which are highly suggestive of acute irritant poisoning. Because of the fact that the symptoms of simple perforating ulcer of the duodenum are in many cases so indefinite—in other words, because of the fact that simple perforating ulcer of the duodenum is in many cases so entirely latent—the differential diagnosis of the sudden rupture of a duodenal ulcer and of acute irritant poisoning might, from the symptoms alone, be impossible during life. An accurate chemical analysis of the vomited matters, if there were vomited matters, might be the only possible means of distinguishing the two conditions during the life of the patient. In cases of ulcer of the duodenum which perforate, the vomiting is usually much less marked than in cases of acute irritant poisoning. This is a point of considerable diagnostic value.

If it were ascertained that a person, who had been suddenly seized with pain in the abdomen, with vomiting, and collapse—symptoms common to acute irritant poisioning, and the sudden rupture of a perforating ulcer of the duodenum —had previously suffered from the symptoms of internal hæmorrhage, and had after the occurrence of such symptoms passed blood by the bowel, or had vomited blood; and that he had previously complained of pain or uneasiness in the region of the stomach, and of dyspeptic symptoms, a positive opinion of disease (ulcer of the stomach or duodenum) might be ventured upon.

From this description, it will be apparent that the symptom above all others which is of real diagnostic value for the diagnosis of simple perforating ulcer of the duodenum, and for the differential diagnosis of the sudden rupture of a perforating ulcer of the duodenum, and of acute irritant poisoning, is a history of internal bleeding, and the passage of blood by the stools. As I have also stated above, the vomiting is, as a rule, much more urgent in acute irritant poisoning than in ulcer of the duodenum which has perforated.

After the above was written (for the paragraph relating to perforating duodenal ulcer was added afterwards, and was not communicated to the students at the clinic), I received a letter from my friend Mr Frederick Page of Newcastle-on-

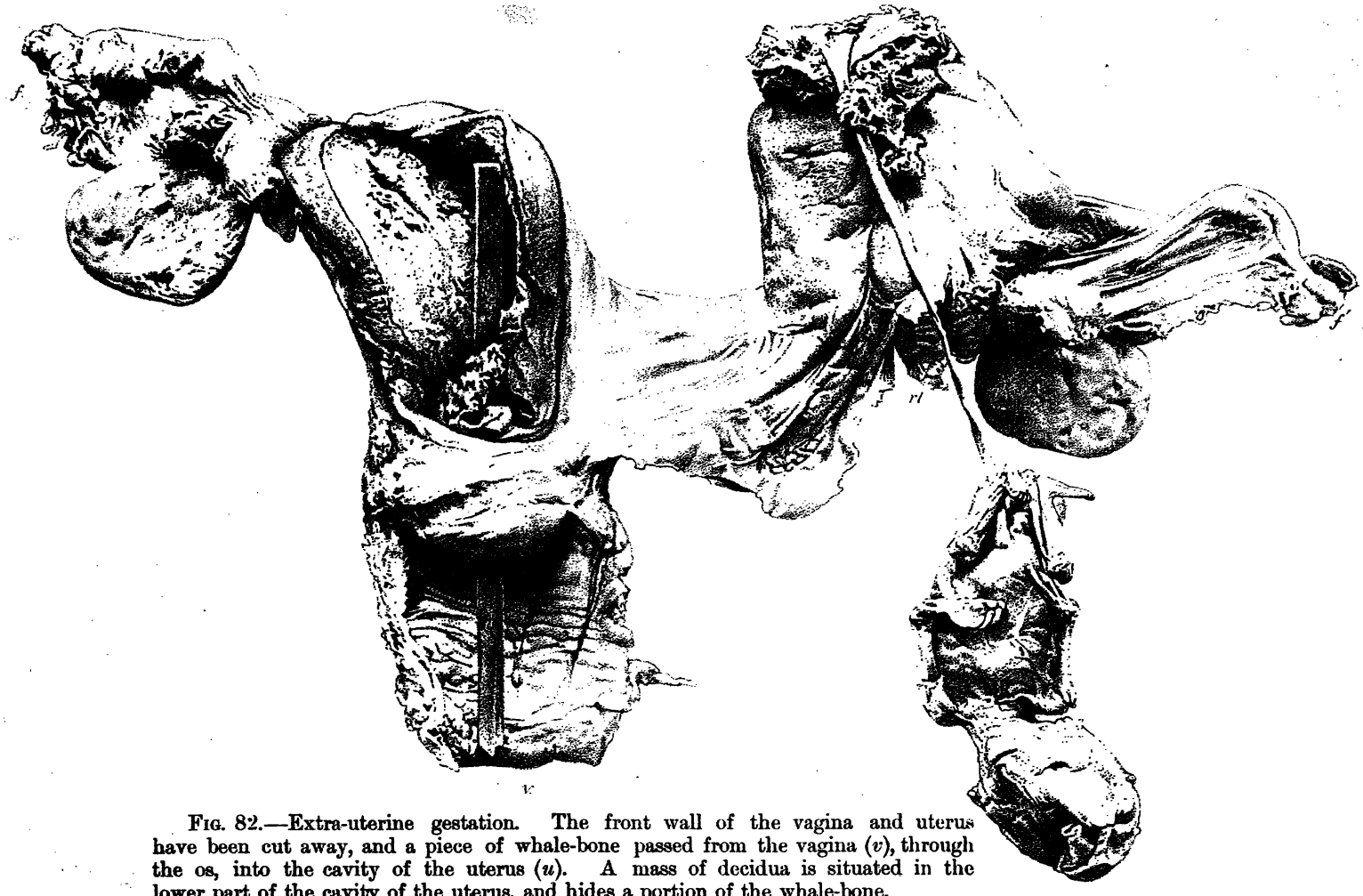


FIG. 82.—Extra-uterine gestation. The front wall of the vagina and uterus have been cut away, and a piece of whale-bone passed from the vagina (*v*), through the os, into the cavity of the uterus (*u*). A mass of decidua is situated in the lower part of the cavity of the uterus, and hides a portion of the whale-bone.

The right ovary (*o*) and the right fallopian tube (*f*) are normal. Between the left ovary (*o'*) and the left fallopian tube (*f'*) on the one hand, and the uterus on the other, a sac, fully the size of a large hen's egg, is situated. The round ligament (*r.l.*) appears to spring from the middle of the sac, which is formed by the dilated fallopian tube, and is completely filled with placenta. The sac has ruptured at its upper surface, and through the rent (which measures $\frac{3}{4} \times \frac{1}{2}$ an inch) that portion of the placenta to which the umbilical cord is attached, is seen to project.

The fœtus is lying in the cavity of the abdomen.

Tyne, in which he says:—"The case of mine you quote was singularly brought to my mind a little time ago. I have seen several others very like it. I got a hasty summons to Middlesboro, and on arrival found the patient dead. He had been seized the day before with acute pain in the pit of the stomach, for which opium was given; he got no relief, and died early the next day. I suggested that there was perforation, but was doubtful as to where. On a post-mortem examination, an ulcer was found to have given way on the deep surface of the duodenum. The man was some 40 years of age, in a good position, and had not been ill."

The rupture of an extra-uterine, or as Mr Lawson Tait terms it, *an ectopic gestation*, may be attended with great pain in the abdomen, collapse, vomiting, and even in some cases diarrhœa. These symptoms may occur so suddenly, and in the midst of such apparent good health, that they may be thought to be due to acute irritant poisoning.

I have in my possession a very beautiful specimen of extra-uterine gestation, in which the suspicion of irritant poisoning was actually entertained. The patient, a young unmarried woman, while dancing at an evening party, was seized with pain in the abdomen. She vomited repeatedly, was taken home and died, some six or seven hours afterwards. My friend Dr Wilson of Wallsend, to whom I am indebted for the specimen, was called to see her, but found her dead. It was thought that she might have been poisoned by something she had eaten at the party. The Coroner was consequently communicated with. At the post-mortem examination, the abdomen was found full of blood; it had escaped from a ruptured extra-uterine gestation. (A drawing of the specimen, which is represented in fig. 82, was made on the black board.) The specimen is a very beautiful one. A piece of whale bone has been passed from the vagina (v) into the cavity of the uterus. The cavity of the uterus is slightly enlarged; it contains, you will see, a small mass of decidua. The right Fallopian tube and ovary are normal. A cyst, fully the size of a duck's egg, is situated apparently in the broad ligament, between the uterus and the fimbriated extremity of the left fallopian tube. The cyst is completely filled with placenta. The membranes in the cyst are *in situ* and undisturbed. The cyst contains no blood; it is completely filled, as I have just stated, with the placenta and its membranes. The fœtus, which measures three inches in length, is lying in the cavity of the peritoneum. It is attached, you will see, to the placenta by the umbilical cord. The ruptured orifice in the cyst is small and circular, it measures one inch by three-quarters of an inch. A small mass of the placental villi, from which the fatal bleeding had taken place, is seen projecting through the rupture into the cavity of the peritoneum.

Now it is obvious that the fœtus was not, at the time of rupture, contained in the Fallopian cyst. The cyst is completely filled with the placenta and the membranes, and the opening in the cyst is so small, that even if the fœtus had been in the cyst, it could hardly have escaped through it.

Possibly a sac of membranes, which was cleared away in removing the clots from the cavity of the abdomen, surrounded and included the fœtus. There is, however, no evidence of this, so far as I can see, in the preparation. Another explanation which has been given me by Dr Hart, who has kindly examined the specimen, is, that the fatal rupture which caused the bleeding may not have been the first rupture. According to this view, the fœtus was for a time contained in the Fallopian cyst; the cyst ruptured at an early stage of the pregnancy; and the fœtus escaped into the cavity of the abdomen; at the time of this first rupture, there was no bleeding; the fœtus continued to grow in the cavity of the peritoneum, and the placenta continued to grow in the cyst from which the fœtus had escaped; ultimately, at the third month of pregnancy, the Fallopian cyst, which was now completely filled with placenta, ruptured a second time, and the fatal bleeding occurred. This seems a very plausible explanation. In any case, the specimen is of great beauty and of considerable interest.

The notes of the case were read at a meeting of

the North of England Branch of the British Medical Association, which was held at Tynemouth, on the 29th of April 1875. The following is the report of the case, which was published by Dr Wilson, in the *British Medical Journal* for June 5th, 1875, page 746:—

"About 5 a.m., on September 9th last, I was sent for," says Dr Wilson, "to see M. A. F., aged 22, who was said to be dying. I knew the girl, and had indeed seen her at my surgery on the previous afternoon, when she came for her father's medicine. She then appeared to be in robust health. When I arrived at the house, I found her dead. She lived with her father, who was a widower, and attended to his household. He gave me the following account of the circumstances immediately preceding her death. She had gone to a dancing party on the previous night, and, after dancing a short time, complained to her partner that she was in pain. He procured her a small quantity of brandy, which relieved her, and she resumed the dance. She was, however, obliged to desist, and sought her home at about eleven, or near midnight. She then suffered from severe pain in the abdomen. Her father, who alone was present during the night, told me that the symptoms were, violent retching and vomiting at intervals, with severe abdominal pain. About 4 a.m., she expressed a desire to have her bowels relieved, and rose for that purpose. After sitting a short time, she fainted, and being removed to bed, died shortly afterwards. I was told by a neighbour that she had not been regular for three months, and had been taking medicines with the object of re-establishing the catamenial functions. The case was so peculiar and suspicious, that I thought it advisable that an inquest should be held. This was done, and I made a post-mortem examination on the evening of the same day, assisted by Dr Huntley of Jarrow.

"As the possibility of poisoning was not overlooked, care was taken to have suitable vessels to contain the necessary viscera. There were no external features deserving of remark. To all outward appearance, the body was that of a well-nourished healthy girl. The os uteri indicated that the uterus was unimpregnated. The abdomen was considerably distended, and at the lower part, emitted a dull sound on percussion. We examined the contents of the cavities of the head and chest, and found all the organs in a perfectly sound and healthy state. On making an incision into the abdomen, however, we were somewhat surprised to find the peritoneum filled with a large quantity of clotted blood, which directed our suspicions to the true nature of the case. During the removal of this blood, a fœtus was discovered floating in it, and a further exploration revealed the Fallopian tube dilated and ruptured, with a placenta protruding through the rent. The case itself is not a very unusual one; indeed, it is perhaps the most common variety of extra-uterine gestation. But its surroundings, and the fact of its occurring in an unmarried woman, who had had no children, invest it with an interest which it seemed to me might be of service from its medico-legal bearing, as an aid to diagnosis in any similar contingency."

Cases of extra-uterine gestation of this description, in which the sac ruptures at an early stage of utero-gestation—usually about the sixth week—and in which alarming symptoms, which may resemble more or less closely those of acute irritant poisoning, are suddenly produced, are by no means very uncommon. I have myself met with one other case, which was not, however, verified by post-mortem examination, in which I am satisfied that the rupture of a Fallopian pregnancy was the cause of death. As Mr Lawson Tait, whose work on this subject deserves, in my opinion, our warmest approbation, has pointed out, that the only satisfactory treatment in such cases is to open the abdomen, pass the hand quickly into the pelvis, feel the position of the uterus, then pass the hand along the broad ligament, first on one side, and then, if the extra-uterine sac is not found on that side, along the broad ligament on the opposite side. If the sac which contains the extra-uterine gestation is not found in connection with the broad ligament which is first examined, it is sure, Mr Lawson Tait states, to be situated in connection with the broad ligament on the other side, for there is no other

known condition—the sudden rupture of an aneurism of one of the large vessels of the abdomen, and perhaps the rupture of a dilated or varicose vein, I should say, alone excepted—in which, in an apparently healthy woman, a large quantity of blood is rapidly extravasated into the cavity of the abdomen, with the production of symptoms which I have just described.

The broad ligament, with which the extra-uterine gestation is connected, should then be drawn out of the pelvis, transfixed and ligatured, and the cyst removed.

At a recent meeting of the Royal Medical and Chirurgical Society of London (reported in the *Lancet* of November 16th, 1889), a discussion took place on the subject of extra-uterine gestation. Considerable difference of opinion seems to have been expressed as to the treatment which should be adopted in cases of ruptured Fallopian gestation. It may, I presume, be granted, as was stated by more than one of the speakers, that the rupture of a Fallopian gestation is not always fatal. But one great authority went so far as to say that "his own impression was that those which were let alone did quite as well as those which were operated upon."

To me, with my comparatively small knowledge of gynæcological matters, this seems a very remarkable statement.

That many women do die from the hæmorrhage into the peritoneum which attends the rupture of a Fallopian gestation, every one must allow. That some of the women who would die can be saved by opening the abdomen and arresting the hæmorrhage, must also, I think, be granted. Further, I presume it is impossible to say in which cases the hæmorrhage will prove fatal and in which a natural arrest will occur.

Now if these propositions be granted, it must I think, be conceded that the practice which Mr Lawson Tait advocates is both logical and correct. The only possible argument against opening the abdomen and endeavouring to arrest the hæmorrhage, under such circumstances, would appear to be that the additional risks which the operation entails—the risks of aggravating the hæmorrhage or of producing subsequent sepsis—are greater than the risks of leaving the patient alone in the hope that the hæmorrhage will be arrested by nature.

Now the risk of sepsis, in the case of an intra-peritoneal hæmorrhage, such as is present under the conditions which we are considering, may, I presume, be said to be very small. (It is necessary to draw a sharp line of distinction between intra-peritoneal and extra-peritoneal hæmorrhage. No one, so far as I know, not even Mr Lawson Tait himself, advocates the opening of an extra-peritoneal hæmatocele. Mr Tait, I believe, distinctly states that such a procedure is not only uncalled for by the urgency of the symptoms, but is actually harmful.)

The risk of aggravating the bleeding by operation and manipulation, and so killing the patient, is, so far as I am able to weigh the evidence and give a judgment on the point, decidedly less than the risk of allowing the hæmorrhage to continue with the hope that a natural arrest may occur.

Further, I would point out that in desperate cases, in which the patient is practically pulseless, and in which the slight additional hæmorrhage which the opening the abdomen and the manipulative interference in the pelvis does presumably favour, we have in transfusion a means of averting this danger.

In the hands of Dr John Duncan, Dr Cotterill, and other surgeons, the injection of saline solutions into the vessels has been proved to be a valuable means of dealing with such desperate conditions, in ordinary surgical practice. If bleeding, which seemed likely to prove fatal, were taking place in any ordinary surgical case, no one would, I presume, be content to wait in the hope that a natural arrest of the hæmorrhage might be effected. Every surgeon would, I presume, under such circumstances, make an effort to expose the bleeding point and to secure the bleeding vessel. If the patient were pulseless, it probably might be advisable, before proceeding to expose the bleeding point, to perform transfusion.

It is now some years since I took any active

part either in surgical or gynæcological practice, but the statements which I have just made represent the subject, so far as my limited knowledge and limited experience enable me to represent it.

At any moment, any one of us, who is engaged in active practice, may be brought face to face with a case of ruptured Fallopian gestation. Rare as the condition is, one can never tell when it may arise. The consulting physician does undoubtedly, though fortunately very rarely, meet with such cases. It is consequently necessary to have some knowledge on the subject, and to be prepared to give some opinion—for in such emergencies it may be impossible to secure an adviser who is in the habit of dealing with such cases.

For my own part, and with the limited knowledge I have of the subject, I unhesitatingly affirm, that if any one in whom I were interested were bleeding into the peritoneum from a ruptured Fallopian gestation, I would, provided that a competent operator could be obtained, advise that the abdomen should be opened, and an attempt, at all events, made to arrest the hæmorrhage by surgical interference.

Although the sudden rupture of an extra-uterine gestation, may resemble in some of its features acute irritant poisoning, the differential diagnosis is not in reality difficult. There is one symptom or sign which is characteristic of the rupture of an extra-uterine gestation, and which is not present in acute irritant poisoning, and that is, the pallor and bloodless condition of the patient. In the vast majority of cases, the presence of marked anæmia would absolutely decide the question. It is, of course, possible that a woman who was suffering from chlorosis or pernicious anæmia might be poisoned. In such a case you would have the symptoms of acute irritant poisoning, and, in addition, marked pallor and anæmia. But in such a case, the history would help us to decide the question. On inquiry, the fact would be elicited that the patient was pale and anæmic before the acute symptoms developed; in other words, that the bloodless condition was not the result of the acute seizure—did not result from the same cause as the abdominal pain and vomiting.

Then again, the anæmia which attends the rupture of the extra-uterine fœtation, is acutely progressive; every now and again the patient faints, or complains of feeling faint, as more and more blood is every now and again extravasted. In many cases of ruptured extra-uterine gestation, the rupture can be distinctly traced to some strain or unusual exertion. In my experience, too, women who are suffering from chlorosis or pernicious anæmia, do not, as a rule, become pregnant, even supposing they are married. The vomiting, too, which attends the rupture of an extra-uterine gestation, is in many cases rather retching than the evacuation of any considerable quantity of stomach contents; while the vomited matters never, so far as I know, contain blood; and the pain and tenderness are referred to the lower half of the abdomen, rather than to the region of the stomach. I repeat, that the profound and rapidly developed anæmia, and the tendency to repeated fainting, are the essential features on which the differential diagnosis has to be based. Irrespective of the other points which I have mentioned, they are usually quite sufficient to decide the question, and to distinguish the rupture of an extra-uterine gestation from acute irritant poisoning.*

The symptoms which attend the sudden rupture of a hydatid cyst connected with the liver, or other abdominal viscus, might also be mistaken for the symptoms of acute irritant poisoning. Sudden pain in the abdomen, vomiting, collapse, and subsequent acute peritonitis, would probably result from the sudden rupture of a hydatid into the cavity of the peritoneum. The same symptoms may be due to the rupture of an ovarian tumour, or of an abscess in the ovary. Very similar symptoms may also, I believe, be

* Some further remarks on extra-uterine gestation were made at this clinic, and the leading particulars of two other cases which had come under Dr Byrom Bramwell's own observation were detailed to the students. As these cases do not bear upon the present question—the differential diagnosis of extra-uterine gestation and acute irritant poisoning—they will be separately recorded in the next number of these *Studies*.

due to the sudden twisting of the pedicle of an ovarian tumour.

It is possible, therefore, that any one of these conditions may be mistaken for acute irritant poisoning. In the case of an abscess in the ovary, a careful inquiry into the previous history would, in many cases, throw light upon the true nature of the case. It must, however, be remembered, that when a woman is collapsed, suffering acute pain, and vomiting, it is not always easy or possible to obtain an exact history of the previous state of health,—to elicit a history of gonorrhœa, of pelvic distress, of pain preceding menstruation, or of menorrhagia. Further, it must be remembered that prostitutes, who are perhaps more liable than any class of women to suffer from gonorrhœal inflammation of the Fallopian tube and ovary, are also perhaps more liable to suffer from violent deaths of all kinds, including acute irritant poisoning, than the majority of the female population. In many cases, however, of abscess of the ovary, which have suddenly ruptured, and are attended with symptoms suggestive of acute irritant poisoning (sudden pain in the abdomen, vomiting, and collapse), a careful inquiry into the previous state of health will show that the patient has previously suffered from symptoms of local pelvic disease.

It is different in the other conditions which I have just mentioned. In a case of hydatid disease of the liver, or cystic tumour of the ovary, which suddenly ruptures, and in the case of an ovarian tumour, the pedicle of which becomes suddenly twisted, there may be absolutely no history suggestive of previous disease. These conditions may have been entirely unattended with symptoms,—entirely latent,—until acute symptoms (pain, vomiting, and collapse) are developed. The differential diagnosis of such conditions and acute irritant poisoning may, therefore, be attended, in some cases, with great difficulty.

The fact that the pain in the abdomen developed after a blow, strain, or violent effort of any kind, would be of importance. If the symptoms did not come on after eating or drinking, acute irritant poisoning would be contra-indicated. The fact that the pain was referred to the abdomen generally, rather than to the region of the stomach in particular, would probably also be of some value. The presence of blood in the vomited matters—granting that the observer was satisfied that the symptoms were not due to the presence of an ulcer of the stomach—would be in favour of acute irritant poisoning rather than of disease. The presence of diarrhœa would be suggestive of irritant poisoning. It must, however, be remembered, that diarrhœa does occasionally, though exceptionally, occur in the diseased conditions which we are considering,—in connection with the sudden rupture of a hydatid or ovarian cyst into the cavity of the peritoneum.

A pelvic examination, in the case of ovarian disease, might, of course, throw some light upon the case.

A careful and judicial investigation into the whole facts of the case (the symptoms and physical signs which are present) will doubtless in most cases enable a well informed and shrewd observer to come to a satisfactory conclusion. But cases do, I believe, every now and again occur in which the diagnosis is attended with so much difficulty, that a positive opinion can only be arrived at by waiting and watching, or by a chemical analysis of the vomited matters.

In order that you may realise how completely latent a hydatid tumour of the liver may be, I will briefly mention the particulars of the following case:—

About fifteen years ago, a sailor was admitted to the Newcastle Infirmary under my care, on account of a lump in the right side, which he stated was due to a blow which he had received the previous day. He stated that he had been perfectly well until the receipt of the injury. He had recently come ashore, and while larking with his companions, as sailors who have just arrived on shore from a long voyage are so apt to do, he had been violently thrown against a post which was sticking upright in the ground. The right side of the abdomen struck against the post. The next day the part was painful, and on examining it he discovered a large lump.

He felt satisfied that the lump was not there before, and not unnaturally attributed it to the injury.

On examining the abdomen, a globular elastic swelling, about the size of an ostrich egg, could readily be detected in the right lumbar region. The swelling was somewhat tender to the touch, and gave distinct evidences of fluctuation. It appeared to be connected with the liver; that organ was very markedly enlarged, the percussion dulness extending high up into the right thorax.

The tumour was diagnosed to be a hydatid of the liver, and the diagnosis was proved to be correct by puncture and examination of the cyst contents—the characteristic hydatid hooklets being discovered. I need not refer to the after progress of the case, which I have recorded in the *Edinburgh Medical Journal*. I mention it now in order that you may realise how completely latent a large hydatid tumour of the abdomen may be. I may say, in passing, that given an *irregular* enlargement of the liver, which is *painless*, unattended with fever, emaciation—in fact, unattended with symptoms—you are (provided that you are satisfied that the condition is not some peculiar congenital malformation), justified in giving a positive opinion that the case is one of hydatid disease.

I may also take the opportunity of emphasising the fact that, in practice, we very frequently find an accidental blow or injury is supposed to be the cause of diseased conditions, which have escaped the observation of the patient, but which have been present long before the receipt of the injury. In the third number of these *Studies*, I referred to this point, and detailed an extraordinary case in which a skilled observer—a medical man—was absolutely ignorant of the presence in his abdomen of a large malignant tumour, fully the size of a child's head.

The symptoms which attend *rupture of the heart, or the rupture of an aneurism at the root of the aorta into the sac of the pericardium*, may, in exceptional cases, resemble those due to acute irritant poisoning.

An instructive case of this description is reported in the *Lancet* for November 9th, 1889, by Dr W. J. Naismyth. The details are as follows:—

"At 6.30 a.m. of May 7th, 1888, Mrs F. B., a field worker, about forty-eight years of age, walked a distance of three miles and a half to her work in the fields. The police information states, that for some years she had had occasional attacks of pain in the chest and breathlessness, but in the intervals was apparently well and fit for her usual employment. At 8 a.m.—that is, an hour and a half after leaving home—she became suddenly very ill, vomited repeatedly, and complained of much pain in her 'belly.' She was taken home in a cart, but medical aid was not obtained till next day. Dr Lawrie, who then saw her, depones: 'I saw deceased at 10.30 a.m., and found her in bed complaining of very severe pain in the epigastric region, but not in the heart. She told me she had been taken ill in the fields the morning before, and had suffered the same pain as she had now; that she had vomited before being brought home, and had done so considerably since (no vomited matters had been preserved); and that she had suffered this epigastric pain all night long, indeed, since ever she took ill. She was in so much agony as to be unable to explain anything. I thought her suffering from inflammation of the stomach or peritoneum. and said so, ordering stimulants, as there was great debility, and poultices to the abdomen. There was nothing noticeable in the character of the breathing. I had hardly left the house, not having gone far, when I was recalled, and was only in time to see deceased expire, after an illness, I understand, of about twenty-six hours. It subsequently occurred to me that the death might have been due to irritant poisoning, though from the absence of marks in the mouth, not by the more usual corrosives, I understood the vomit had all gone on the floor. In short, a certificate of death from natural causes being, in the circumstances, properly refused, I was instructed, by the order of the Procurator Fiscal, to make, in conjunction with the medical attendant, whose precognition I have just quoted, a post-mortem examination.

"The following are detailed excerpts from the

report, selected solely in respect of their relevancy, and irrespective of their order of sequence in the body of that document:—

"'The body, which was that of a female about 50 years of age, was well nourished, bordering on obesity. . . . There were no marks of vomiting, or staining of the lips, tongue, or hands. . . . The stomach and gullet, which were ligatured and removed for separate examination, along with the upper portions of the small intestines, were all empty, healthy, and normal, exhibiting no erosion or inflammatory action on their lining surfaces. Some adhering mucus in the stomach had the usual acid, but no other distinctive odour. . . . The other abdominal organs and the peritoneum were healthy and normal. . . . On opening the chest, the pericardial sac was observed to be livid and tensely distended with large clots and dark fluid blood, which entirely surrounded and compressed the heart. The effusion was discovered to have found exit through a minute opening in an aneurism of the size of a bantam's egg, involving the base or commencement of the aorta, that portion of it, namely, which is situated within the pericardial precinct. The aneurism occupied the front and lateral portion of the vessel, but may be considered to be of spurious, diffuse, or consecutive type; for by a process of ulcerative thinning, an opening of the size of a threepenny piece communicated with a sacculated condition of the corresponding portion of the pericardium itself. The outer or cardiac wall of the tumour, therefore, consisted of the pericardium only; and it was found that, on its anterior aspect, a minute opening, not larger than a single grain of rape seed, was the point where the hæmorrhage had gained access to the pericardial cavity. The aorta exhibited the usual appearances of atheromatous disease. . . . The heart was of normal size, but pale, soft, and friable; its right and left cavities almost empty and partially contracted, its valves fairly competent; pericardial serosity, of course, ungaugeable. . . . Opinion: From the foregoing examination of the body of the said F. B., we are of opinion that death was caused by the rupture of an aortic aneurism; and, from the small size of the rupture, that death did not occur suddenly, but after the lapse of some hours.'

The severe pain, vomiting, and collapse, which may result from *the passage of a gall stone*, and in some cases of *a renal calculus*, may resemble more or less closely the symptoms of acute irritant poisoning.

The locality and character of the pain, the fact that the vomiting is not as a rule urgent (in many cases nausea rather than much vomiting), and especially the colour of the vomited matters, when taken in conjunction with the history of the case (in many cases, attacks of a like kind having occurred before), would usually enable a correct diagnosis to be made without any great difficulty. The after progress of the case, and the development of jaundice in the case of a hepatic colic, and of hæmatemesis in the case of renal colic, are of course conclusive.

In some cases in which *obstruction of the intestine is suddenly produced by a twist or kinck of the gut, a volvulus or an internal strangulation of any kind*, the symptoms may be thought to be due to acute irritant poisoning.

A very remarkable case of this description came under my observation some three years ago. On November 8th, 1886, I was asked by my friend Dr George Mackay, to see a boy, aged 12, who had suddenly been seized with pain in the abdomen and vomiting, and who was dying from symptoms which, it was thought, might perhaps be the result of acute irritant poisoning. The suspicion of irritant poisoning was largely based on the fact that the boy's mother had also been seized, some seven or eight hours after the lad had become affected, with pain in the abdomen, vomiting, and diarrhœa.

On arriving at the house, I found the boy in a dying condition—collapsed, the face and hands blue, the radial pulse imperceptible, the heart beating 170 times a minute. The abdomen was only slightly distended, but notwithstanding the collapsed condition of the patient, exquisitely tender to the touch; the external temperature was not elevated; there had been no vomiting for several hours. In the condition in which the boy was, any lengthened examination would have been cruel, and it

was not made; we contented ourselves with the administration of stimulants, and doing what we could for the patient. As a matter of fact, he died some five or six hours after the consultation.

On inquiring into the facts of the case, I learned that the boy had been well until three days previously. On the morning on which he was taken ill (Saturday, November 5th), he had gone out for a run before breakfast, as he was accustomed to do; while running, he had complained of pain in the abdomen, and had been obliged to come home; as soon as he got into the house, he vomited. During the course of the day the pain in the abdomen and the vomiting had continued, and the boy had complained of being thirsty; the vomited matters were of a dark brown or black colour. In the course of the morning, Dr Mackay was sent for; he found the patient complaining of great pain in the abdomen, which was only moderately distended, and vomiting repeatedly, the vomited matters being of a black colour, and evidently containing blood; the bowels had been freely opened since the attack commenced; the patient was very thirsty.

Opium and a dose of calomel were prescribed, and fomentations were applied to the abdomen. The patient did not, however, improve; the pain, vomiting, and thirst continued; the temperature became elevated. There was never any great distention of the abdomen. The case went on from bad to worse, until the time of my visit, when the patient had obviously only a few hours to live.

Now if the case had stood by itself the suspicion of acute irritant poisoning would probably not have been entertained; the history of the case, the way in which the symptoms had developed, and the symptoms themselves —with, perhaps, the single exception of the vomiting of black stuff which resembled coffee grounds, and was supposed, and rightly supposed, to be blood—were in favour of disease (acute obstruction of the intestine) rather than of irritant poisoning. The distention of the abdomen was not, however, so great as one generally sees in acute intestinal obstruction, but this could perhaps be accounted for by the very incessant and urgent vomiting, which was quite exceptionally severe and frequent in the early stages of the attack.

The fact which raised a suspicion of poisoning was this, that the boy's mother, who had her supper with him the night before he took ill, was also seized with pain in the abdomen and vomiting some seven or eight hours after he had himself become affected.

At the time I visited the boy, the mother was still suffering, and was confined to bed.

In her case, the symptoms were not very urgent; there had been some pain and tenderness in the region of the stomach; she had vomited some half dozen times; the vomited matters did not, however, present any appearances worthy of note.

At the time of my visit, there was no longer any pain or tenderness on pressure in the abdomen; the vomiting had ceased, though she still felt inclined to be sick; her face was flushed; her tongue furred; pulse quick—120 in the minute; the temperature 102° F. The vomited matters in the mother's case had not been of a black or brown colour.

Now the fact that both the mother and the boy had been seized with pain in the abdomen and vomiting was very suggestive that the symptoms were the result of some form of irritant poisoning.

A careful investigation into the collateral circumstances of the case tended, however, rather to negative than to establish this view.

It appeared that the day before the patients took ill (November 4th) was the boy's birthday. He had spent the afternoon at a relative's house, and had eaten a bun coated with red sugar. On his way home at night, he had got wet through. It was thought that possibly the sugar, which coated the bun, might have contained some poisonous material; but this view was contra-indicated by the fact that the boy had eaten the whole bun himself; his mother had not tasted it.

After coming home the boy had had supper with his mother; they had eaten the same articles of food, among other things a rich plum pudding. It seemed probable therefore that if

they had been poisoned, the irritant, whatever it was, had been taken at this meal—the last that the lad had had before he took ill.

After eating his supper the boy had gone to bed; had slept soundly, or at all events had not made any complaint either during the night or next morning when he got up. The mother, too. had passed a good night, and had not felt any uneasiness until some hours after the boy had been taken ill.

The length of time that elapsed between the last meal and the onset of the symptoms was strongly against poisoning by any ordinary metallic irritant. It is true that arsenic, when given in the solid form along with food, may not produce immediate effects; and that in such cases the influence of sleep may still further retard the development of symptoms. But the interval in this case was too long, and the absence of symptoms during that long interval was so complete, that the supposition of arsenical poisoning seemed definitely negatived.

If the symptoms were actually the result of irritant poisoning, the most likely supposition seemed to be that the food itself, probably the plum pudding, had acted as the irritant.

Against this view, however, was the fact, which was definitely established on careful inquiry, that the other members of the family —the lad's brothers and sisters—had all eaten freely of the same articles, including the plum-pudding, and that none of them had suffered. The mother, it appeared, had waited till the boy came home to take her supper with him; but the mother and the boy had not eaten of anything which had not been eaten by the other members of the family.

The facts of the case were therefore very puzzling; and when the boy died we looked forward with much interest to the results of the post-mortem examination.

The post-mortem examination was made on November 10th, at 4 P.M. (thirty-six hours after death).

The abdomen was in an advanced state of decomposition. In moving the body from the bed to the table on which the examination was made, some brown fluid escaped from the mouth.

The abdomen contained 6 oz. of blood-stained fluid; the peritoneum was acutely inflamed; a large coil of the small intestine was gangrenous its walls were so friable that, in turning it gently over, it ruptured, and some blood-stained fœcal matter escaped into the cavity of the peritoneum.

The portion of gut which was gangrenous had passed through an opening which existed in the mesentery, and had become constricted by a dense band of fibrous tissue.

The stomach contained two ounces of brown fluid; the mucous membrane was here and there stained of a brownish colour; the staining was arranged in a streaky fashion; the patient had been taking bismuth, and it was thought that the brown discolouration of the stomach was perhaps, in part at least, due to this cause. In places the stomach was congested, but not obviously inflamed; the cardiac end was softened by post-mortem changes.

The duodenum contained some brown fluid; its mucous membrane was healthy. There was no evidence of inflammation in the small intestine except in the portion of the bowel which was strangulated.

The conditions of the stomach and intestine were not suggestive of acute irritant poisoning.

Farther, I satisfied myself that there was no arsenic in the stomach or intestine.

The case of this boy, when taken in combination with the fact, that the mother was affected in a similar manner to the boy, is a very remarkable one.

The most likely theory to account for the whole facts of the case, appears to me to be the following: The food, probably the plum pudding, acted as an irritant to the mother and the boy who died, but did not cause symptoms of irritation in the other members of the family. The symptoms of gastro-intestinal irritation first showed themselves in the boy, while he was out for his morning run; the small intestine at the same time, or perhaps shortly afterwards, was forced, by the violent peristalsis occasioned by the irritation and colic, through the opening in the mesentery, and became constricted by the fibrous band, to which I have already referred.

The cause of death was obstruction of the bowels, and not irritant poison.

The feature in the case which is chiefly interesting from a medico-legal point of view, was the fact, that the mother and boy were both affected with symptoms indicative of irritant poisoning, and that, in the boy's case, the gastro-intestinal irritation was complicated with intestinal obstruction, and had apparently contributed to the production of that obstruction in the manner which has been suggested above.

On the theory which I have advanced, the mother, like the boy, actually suffered from acute gastro-intestinal irritation.

Another theory which suggested itself to my mind, as an explanation of the occurrence of symptoms both in the mother and the boy, was, that the symptoms in the mother were the result of mental or neurotic causes; in other words, that the mother, being anxious and distressed by the illness of the boy, herself manifested the same symptoms from which he was suffering. A little consideration, however, convinced me that this view was untenable, for the following reasons:—The mother was by no means a nervous, emotional, or hysterical person; although vomiting, increased frequency of pulse, pain in the abdomen, and possibly even tenderness on pressure over the abdomen, might have been the result of mimicry, the very distinct elevation of temperature (102° F.) which was present, was not likely to have been produced in this manner. The whole impression left on my mind, after a careful investigation and consideration of the whole facts of the case, was that the symptoms in the mother's case were undoubtedly the result of a definite local irritant.

The *onset of scarlet fever, small-pox, epidemic cerebrospinal meningitis, and doubtless other acute febrile diseases,* is occasionally in children attended with symptoms which resemble more or less closely those of acute irritant poisoning.

Soon after I entered general practice, I had the opportunity of seeing a very widespread epidemic of scarlet fever, in which the type of the disease was unusually severe. During the course of that epidemic, I met with more than one case in which the patients were within a few hours overwhelmed by the severity of the scarlet fever poison. Two of these cases occurred in children between the ages of one and two years. In both cases, the attack was ushered in by severe vomiting and diarrhœa, followed by collapse, and death within twenty-four hours; in one case, there were convulsions at the onset. In neither case did the eruption shew itself, and on account of the age of the patient, the characteristic sore throat was not complained of.

So far as the symptoms in these cases went, it would, I believe, have been impossible to come to a definite diagnosis as to the nature of the illness. The true character of the attack was, however, in both cases, demonstrated by the fact that, either before or after these children were attacked, other grave cases of scarlet fever appeared in the brothers and sisters of the patients.

On the 19th November 1872, I was asked by the Coroner for South Northumberland to make a post-mortem examination on the body of a boy, who was taken ill, and died at Whitley, under the following circumstances*:—

Deceased, who was eleven years of age, got up on the morning of November† 16th, 1872, as usual. He seemed well and cheerful, and made no complaint. He was sent to a neighbouring house for some milk for breakfast; after an absence of a few minutes he returned, and took a drink of the milk; immediately he said he felt sick, and vomited freely; he continued to feel sick and giddy, and was put to bed. When his father came home to dinner (he had no mother, the father and the boy living alone in the house), at twelve o'clock, he found the lad complaining of sickness and thirst, he was very

* I quote from the report of the case which I published at the time in the Transactions of the Northumberland and Durham Medical Society. The leading particulars of the case were mentioned to the students at the clinic.

† In the report, which is printed in the Transactions of the Northumberland and Durham Medical Society, the case is stated to have occurred on the 16th of December. This is obviously a mistake, for the paper was read on December 12th.

feverish, but did not appear to suffer pain. As the day went on the boy got worse; in the evening he became unconscious, and continued so all night. He was seen on the morning of December the 17th, by the Vicar of Whitley; (a doctor had been sent for, but, through a misunderstanding, did not attend). He was then in a state of coma, the breathing stertorous, there were convulsive twitchings of the muscles of the face and extremities, the skin was very hot and dry, and there were several livid discoloured patches on the face. He died at 4 p.m., on December 17th. For half an hour before death he was severely convulsed.

These facts, together with the circumstance that the patient was of an eccentric disposition, led to the belief that he had been poisoned by something which he had taken in the milk,* a supposition which was proved to be quite unfounded by the following facts:—*Firstly*, the remainder of the milk was drunk by the father without any bad effect; *Secondly*, there were no post-mortem appearances to indicate poisoning, and there were most marked appearances the result of disease.

A post-mortem examination was made thirty-six hours after death. The body was very thin; externally there were no marks of violence. There were several small cicatrices on the legs and thighs (the result of old abscesses, apparently connected with the bone), and well marked opacities of each cornea. Internally the stomach was empty; the mucous membrane of the cardiac end was softened, and in parts stripped off from the subjacent tissue. This change was evidently post-mortem, owing to the action of the gastric juice. There was no congestion or inflammation. The intestines were congested in one or two places, but not to any great extent. Both kidneys were extensively diseased, more especially the right one, which was enlarged, softened, and completely disorganised; it looked like a mass of bloody pulp. The left kidney contained a large cyst; it was less disorganised than the right. The bladder was empty, its walls were very much thickened, especially the mucous coat, which was soft and friable, and in places detached. The other abdominal organs were natural. The left pleura was firmly adherent in its upper half. The lungs were normal. There was a large white patch on the anterior surface of the heart. The heart itself was normal. The brain was soft, but otherwise healthy. (There is no statement in this report as to the condition of the throat; and at this length of time I cannot trust my memory sufficiently to supply the deficiency.)

On inquiry, I ascertained that the deceased had been a very delicate boy from birth, and that, for several months before his death, he had been unable to retain his water, which was scanty, and, at times, entirely suppressed.

As the result of this examination, I came to the conclusion that extensive renal disease had been going on for some time, and that deceased had died from uræmia.

I subsequently learned that a child had died at the milkman's house the very day the boy took ill. I was told that this child had died of ulcerated sore throat. This threw additional light upon the case. The milkman's child had probably died of scarlet fever, for children rarely die (I should now say, do not die), of simple ulcerated sore throat. The boy had either inhaled the poison of scarlet fever when he entered the house, or less probably (I now think more probably), had drunk it in the milk. The scarlet fever poison at once attacked the previously diseased kidneys, causing the sudden appearance of the symptoms, and leading to the rapid termination of the case.

In reply to a criticism raised by a gentleman at the meeting to which this paper was communicated, I stated that I based my opinion that the milkman's child had died from scarlet fever upon the probabilities of the case. Death from simple ulcerated sore throat was so exceedingly rare, and diphtheria was such a very uncommon disease (I referred to the occurrence of diphtheria in that particular neighbourhood—it was exceedingly rare in my practice), that the probability of the milkman's child having died from either of these diseases was small—very small when compared with the probability of scarlet fever.

* I mean some inorganic, chemical poison.

I should have added that scarlet fever was epidemic in the neighbourhood at the time when the case occurred. I may also add that the fact that the patient was very feverish is distinctly opposed to death being due simply to uræmia.

On looking back on the case, and reviewing all the facts in the light of extended knowledge and experience, I am disposed to adhere very strongly to the opinion advanced at the time when the case occurred, and to think that the patient died from the effects of an attack of scarlet fever; the rapidity of the fatal result and the character of the attack being largely due to the disease of the kidney which was previously present.

In connection with the differential diagnosis of acute irritant poisoning and the onset of acute contagious diseases, I must not omit to refer to *Asiatic cholera.*

The symptoms which attend the onset of a bad case of epidemic or Asiatic cholera may very closely resemble those due to acute irritant poison. The first case of Asiatic cholera which came under my own observation might very readily have been mistaken for acute irritant poisoning had not the disease been known to be epidemic. The particulars of that case are as follows:—

One morning during the early part of October 1866 (I do not remember the exact date), a message arrived for my father, while we were seated at breakfast, to go and see a man who had been taken ill at some large engine works, which were situated at a short distance from our house. Being a second year's medical student, at home for the vacation, I was sent to see the case. I found a man lying on a bench in a state of profound collapse. Soon after going to his work, at six o'clock, he had, it appeared, been seized with violent vomiting and diarrhœa. These symptoms had continued at intervals, until the time of my visit. At eight o'clock, when I arrived, the patient was almost pulseless; his features were pinched; the surface of his body cold, of an ashy grey colour; the lips, nose, and finger-tips blue; and the forehead covered with a clammy sweat; his voice was so weak and husky that it was almost imperceptible. He urgently demanded water; and he complained of pains and cramp in the legs. I remember the man's appearance and whole surroundings to-day, as vividly as if he were now before me. It was obvious even to me—young and inexperienced as I was—that the patient was dying. I accordingly hurried back home. My beloved father, who was without exception the ablest all-round practitioner I have ever met, and who had previously seen two severe epidemics of the disease, on hearing my description of the case, at once said that it was in all probability a case of Asiatic cholera. Several cases of the disease had occurred in some of the surrounding villages in Northumberland, but this was, so far as I know, the first case in our immediate neighbourhood. On visiting the patient, my father found that his suspicions were only too correct; he predicted that we were probably at the beginning of an epidemic; and such was, in fact, the case. During the next three or four weeks, the disease raged with terrible severity; many hundreds of persons were attacked in North Shields and Tynemouth. I find it stated in the reports of the Northumberland and Durham Medical Society (November 8th, 1866, page 23), that during the month of October there were 116 fatal cases of the disease in North Shields and Tynemouth, the population being, I suppose, at that time, somewhere about five-and-thirty thousand. The people were panic-stricken. The authorities of the town undertook to supply medical attendance to all those persons who were too poor to pay for medicines or medical advice. The poor people in the town were free to send for any doctor they might think fit; and my father, who was revered and respected by all classes of the community, was literally overwhelmed with work. I shall never forget the fortnight during which the epidemic was at its height. The strain and nervous excitement were to me,—a young lad, unaccustomed to see disease and death,—something terrible. During the ten days or fortnight that the epidemic was at its worst, my father and myself were rushing about from early in the morning till late at night. I do not think that there was a

night during the whole of the time that we were not called out of bed—sometimes many times the same night—to visit some one who had been suddenly attacked with the disease. Such deathbed scenes as I saw during that epidemic, I fervently trust that I may never again witness. At the end of the epidemic, I was myself attacked with choleraic diarrhœa; fortunately the attack was a slight one, and was arrested by appropriate treatment.

My present object is not, however, to record the experiences of that, to me, eventful period, or to give a clinical description of Asiatic cholera. Possibly on some future occasion I may return to these points. I have introduced the subject here with the object of pointing out how closely some of the symptoms of a severe attack of epidemic cholera may simulate the symptoms of acute irritant poisoning.

The patient, whose case I have briefly recorded above, died within a few hours. As is not unfrequent at the beginning of an epidemic, the case was a malignant one. The man was dead within ten hours from the commencement of the symptoms. We subsequently learned that he had been attacked with vomiting and diarrhœa at an early hour of the morning. He had gone to bed feeling perfectly well the night before, and had been able to go to his work at six o'clock. By mid-day he was dead.

I remember my father pointing out how easy it would be to mistake such a case for acute irritant poisoning. Further, I remember him telling me that the essential points for differential diagnosis were the absence of any marked pain and tenderness on pressure in the abdomen, the watery character of the evacuations, and the fact that cholera was known to be epidemic in the neighbourhood.

During the course of that epidemic he referred more than once to the differential diagnosis of cholera and acute irritant poisoning. I remember his emphasising the fact that, during an epidemic of Asiatic cholera, when the people are panic-stricken and the doctors so terribly over-worked as we were during the epidemic of 1866, poisoning by arsenic or corrosive sublimate might very easily be overlooked, and be allowed to pass undetected.

As I have already pointed out, there is not as a rule, in Asiatic cholera, the severe pain and abdominal tenderness which are such characteristic features of acute poisoning by arsenic, corrosive sublimate, and the other metallic irritant and corrosive poisons. Pain in the abdomen may, it is true, be a prominent feature in Asiatic cholera, but, when severe, it is usually intermittent, of a colicky character, and often due to spasmodic contraction of the abdominal muscles rather than to inflammation of the stomach or intestines, and is relieved rather than increased by external pressure.*

The pain, too, in Asiatic cholera is usually developed *after* the vomiting and diarrhœa; whereas in acute irritant poisoning the pain and abdominal tenderness precede, or, at all events, are developed simultaneously with these symptoms.

As a rule, too, in Asiatic cholera, the character of the vomited matters is different from the character of the vomited matters in acute irritant poisoning. In Asiatic cholera, the matters which are expelled from the stomach and intestine are usually more copious and watery, while in cases of acute irritant poisoning, the vomited matters are usually brown, black, or bloody, and the diarrhœa is much more apt to assume a dysenteric character. The rice-water character of the matters evacuated from the stomach and intestines is a characteristic feature of most cases of Asiatic cholera.

The epidemic prevalence of Asiatic cholera, is obviously a point of the highest diagnostic importance, as is abundantly shewn by the case of the patient which has been detailed above.

In acute irritant poisoning, the vomiting precedes the diarrhœa; whereas in Asiatic cholera, the reverse is usually the case, the diarrhœa being in most cases, some writers say in all cases, developed before the vomiting.

In acute irritant poisoning, the suppression

* During the typhoid condition (secondary fever), which sometimes follows the acute stage of Asiatic cholera, abdominal pain and tenderness due to inflammation of the intestine may be present.

of urine is seldom so marked as it is in severe cases of Asiatic or epidemic cholera.

In doubtful cases, the chemical and bacteriological examination of the evacuated matters would be an important point. The presence of the comma bacillus would decide the case in favour of Asiatic cholera; while the presence of arsenic, or corrosive sublimate (provided, of course, these substances have not been given in the form of medicines), would enable one to establish the diagnosis in favour of acute irritant poisoning.

Poisoning by the corrosive acids and alkalis could hardly be mistaken by any intelligent observer for Asiatic cholera. The differential diagnosis of Asiatic cholera, and the other forms of irritant poisoning (other than arsenic and corrosive sublimate), with which it may be confounded—such as poisoning by fungi, decomposing meat, strong purgatives, such as croton oil and elaterium—need not be referred to in detail.

It must not be forgotten that during an epidemic of Asiatic cholera several members of the same family may be simultaneously attacked by the disease. Under such circumstances a suspicion of poisoning might be entertained, more especially if the medical man who was called to attend the patients had not had any previous experience of the disease.

The two conditions could, however, be readily distinguished by attention to the points of differential diagnosis which have been detailed above. Taylor, in his "Principles and Practice of Medical Jurisprudence," gives an instance in which the mistake actually occurred. He says:—"It was just now remarked that there is no disease resembling poisoning which is likely to attack several healthy persons at the same time and in the same manner. This is undoubtedly true as a general principle, but the following case will show that mistakes may occasionally arise even under these circumstances. It occurred in London during the prevalence of malignant cholera in the year 1832. Four of the members of a family living in a state of great domestic unhappiness sat down to dinner apparently in good health. Some time after the meal the father, mother, and daughter were seized with violent vomiting and purging. The evacuations were tinged with blood, while the blueness of the skin, observed in cases of malignant cholera, was absent. Two of these persons died. The son, who was known to have borne ill-will against the father and mother, and who suffered no symptoms on this occasion, was accused of having poisoned them. At the inquest, however, it was clearly shown by the medical attendant that the deceased persons had really died of malignant cholera, and that there was no reason to suspect that any poison had been administered to them. In this instance it will be perceived that symptoms resembling those of irritant poisoning appeared suddenly in several individuals in perfect health and shortly after a meal. We hereby learn that the utility of any rules for investigating cases of poisoning depends entirely on the judgment and discretion with which they are applied to particular cases."*

In severe cases of sporadic cholera—the ordinary summer cholera of this country—the points which have been just emphasised, in speaking of the differential diagnosis of Asiatic cholera, and of acute irritant poisoning (more especially the absence of localised pain and tenderness in the abdomen, the character of the vomited matters, and of the matters evacuated by the bowel), are usually sufficient to enable a well-informed and judicially minded observer to come to a definite conclusion as to the true nature of the case, *i.e.*, to decide whether the symptoms were due to disease (summer cholera), or to acute irritant poisoning (arsenic, corrosive sublimate, &c.)

It might be difficult or impossible to distinguish a severe attack of ordinary sporadic or summer cholera, from a case of poisoning by fungi or other non-metallic irritants. As a matter of fact, summer cholera is very frequently due to eating putrid, decomposed, or indigestible articles of food; the two conditions are therefore practically identical.

* Principles and Practice of Medical Jurisprudence, vol. i., page 192.

Studies in Clinical Medicine.

FRIDAY, DECEMBER 13, 1889.

CONTENTS.

I.—Notes of a Case of Extra-uterine Gestation, in which the Fœtus was Expelled per Rectum.

Mr Lawson Tait and Dr Berry Hart have shown that in some cases of Fallopian pregnancy, in which the extra-uterine fœtation ruptures into the folds of the broad ligament, and in which the bleeding, if any, is consequently extra-peritoneal, the fœtus is ultimately expelled *per rectum*, a communication being formed between the extra-peritoneal sac or cavity, which contains the fœtus, and the lower part of the intestine.

In the following case, which came under my notice in the year 1874, a five months' fœtus was ultimately expelled per rectum. Whether the gestation was originally extra-uterine or not, it is perhaps impossible to decide. But that such was the nature of the case seems to me, with my now rusty and limited knowledge of gynæcology, the most probable supposition.

The notes of the case were read at the same meeting of the North of England Branch of the British Medical Association, at which Dr Wilson brought forward the interesting case of Fallopian gestation which I have published in the last number of these *Studies*. As the case has never been previously published, I take the present opportunity of placing it on record.

The history is as follows:—On the 28th of November 1874, I was asked by my brother, Dr J. W. Bramwell, who had succeeded me as Medical Officer to the Tynemouth Union Workhouse Hospital, to see a patient in that Institution who was suffering from an obscure abdominal tumour.

By occupation a hawker, and thirty-seven years of age, she had been married seven years, but had never had either a child or a miscarriage.

The previous history was as follows:—With the exception of an attack of facial paralysis ten years previously, and of facial erysipelas one year previously, she had enjoyed excellent health until the 26th of April 1874.

On that date (26th April 1874) she was severely assaulted by her husband; two of her ribs were broken, and she was kicked in the lower part of the abdomen and in other parts of the body.

In consequence of these injuries, she was confined to bed for three days. She then got up, and was able to go about, but with difficulty, for a month. During the whole of this period, she suffered from pain in the lower part of the abdomen. At the end of a month, the pain in

the abdomen became so severe that she had again to take to bed. The pain was felt in the lower part of the abdomen, just above the left groin. The belly at this part was, she stated, hard, but there was no distinct lump or tumour.

At this time (the end of May), she menstruated, but the discharge was black and unnatural.

She had menstruated a week before the assault; previous to that time, she had always been regular.

During the whole of June, she was confined to bed. About the beginning of June, she began to vomit. The vomiting generally occurred in the morning; and the vomited matters consisted of the food ingested.

In July, she noticed a lump the size of a hen's egg in the lower part of the abdomen, just above the left groin.

Since it was first noticed, this lump had gradually become larger and more and more painful.

In the middle of September, she again saw a coloured vaginal discharge, and it had continued more or less constantly until the date of my seeing her (November 1874).

The vaginal discharge was scanty in amount, thick and black, almost, she said, like tar.

Since her illness commenced, she had become very weak and emaciated.

On examination I found that the patient was very thin, emaciated, and anæmic.

The abdomen was considerably but uniformly swollen. A brown line of discolouration extended from the pubes to the umbilicus.

On palpation, a tumour was felt in the lower part of the abdomen; it was centrally situated, and extended upwards to a point an inch above the umbilicus, laterally into each lumbar region, and downwards into the cavity of the pelvis.

The upper border of the tumour, which was rounded, but distinctly notched in the middle line, extended rather higher on the left than on the right side.

The part of the tumour which was situated to the right of the middle line was of firm consistence, while the part on the left of the middle line was soft and fluctuating.

Indistinct pulsation, apparently communicated, could be felt in the soft, fluctuating portion of the tumour.

The slightest pressure over any part of the tumour caused great complaints of suffering; the pain was stated to be most intense immediately above the pubes.

She stated that she had constant pain in the region of the umbilicus, and that for some time past she had been obliged to take large opiates for its relief.

There was no pain or tenderness on pressure in any part of the abdomen except over the tumour itself.

The tumour appeared to be fixed, but manipulation caused so much pain, that this point could with difficulty be determined.

The portion of the abdomen which was occupied by the tumour, gave a dull, while the other portions of the abdomen gave a resonant note, on percussion.

No bruit or fœtal heart-sounds could be heard over the tumour.

On vaginal examination, the neck of the uterus was found to be soft and pendulous, and fully an inch in length. The os was very small—a mere depression in the pendulous and papilla-like projecting cervix.

High up in the pelvic cavity, a firm, dense, smooth mass could be felt—presumably the body of the uterus, for the cervix appeared to be continuous with it. This mass was just within reach of, but could not be distinctly defined by, the finger. The slightest pressure on this mass caused complaints of pain.

A uterine sound was passed into the os and through the cervix, but could not, by gentle manipulation, be passed beyond it.

There was no swelling in the pouch of Douglas.

On rectal examination, the firm, dense, painful mass, described above, could be felt high up in the pelvis.

The tongue was clean and moist, but pale and flabby; the appetite poor; the bowels were regular, and the stools natural in appearance.

The organs of respiration, circulation, and urination were normal. The liver and abdo-

minal organs, leaving out of account the uterus and its appendages, appeared to be normal.

The radial pulse numbered 96 in the minute, and was small and weak. The temperature in the axilla was 98.5° Fah. The patient stated that she slept badly; often broke out into a sweat, and sometimes shivered towards morning.

The skin and mucous membranes were pale and markedly anæmic. The muscle soft and flabby, and the body generally much emaciated.

The diagnosis was most difficult. Three views suggested themselves to my mind, viz.: (1) was the tumour connected with the uterus; (2) was it ovarian; and (3) was it a hæmatocele?

The grounds for coming to a positive conclusion, even as regards this preliminary step in the diagnosis, seemed wanting.

In favour of the tumour being uterine, were its position and its apparent continuity with the cervix. But if uterine, what was its nature? It certainly contained a large quantity of fluid, and could not therefore be a fibroid. If the patient's statement could be relied upon, in all probability, it was not the tumour of pregnancy, for she stated that she had had no connection since the assault (April 26, 1874), that she had menstruated naturally a week before that date, and that she had again menstruated, though unnaturally, four weeks after the assault. The history of localised injury to the lower part of the abdomen, the condition of the os and cervix, and the absence of fœtal heart-sounds, or placental souffle, were all against pregnancy. At the same time, the arrest of the menses from May to September, the morning sickness, and the gradual appearance of an abdominal tumour, which appeared to be connected with the uterus, might perhaps be regarded as suspicious circumstances. The fact that there had constantly been, since the month of September, a black tar-like vaginal discharge, seemed to show, granting that the patient had been pregnant, that the death of the fœtus had taken place.* Cancer of the uterus seemed to be excluded by the condition of the os and cervix, for although the new growth is in some cases of carcinoma limited to the body, or even the fundus of the organ, yet the pendulous soft and projecting character of the cervix, and the condition of the os which has been described above, seemed totally opposed to the case being one of cancer. Again, the very evident fluctuation in a portion of the tumour, the great pain and tenderness, the marked emaciation, and the rigors, also seemed in favour of suppuration, rather than of a new growth.

Some of the physical signs of ovarian disease were present, but the history, and the facts of the case, when taken as a whole, left a decided impression on my mind that the tumour was not ovarian.

The history of injury, the evident fluctuation, the extreme tenderness on pressure, and the very marked anæmia, which were present, seemed to favour the diagnosis of hæmatocele. But if the tumour were a hæmatocele, it certainly was not pelvic; this seemed definitely established by the vaginal examination.

On December 6th, my friend Mr Page met my brother and myself in consultation, and the patient was again carefully examined.

On this occasion I again passed a sound into the os uteri; and with a slight amount of gentle manipulation, but without using any degree of force, I succeeded in passing it through a soft stricture, apparently the internal os. Once the sound was introduced into what seemed to be the cavity of the uterus, it passed upwards and onwards with the greatest ease, and its point was soon felt and seen to project immediately beneath the integuments of the abdomen, at a spot two inches immediately above Poupart's ligament, on the right side.

It was evident that the point of the sound was outside the cavity of the uterus. Whether it had passed through a dilated Fallopian tube, or how it had got where it did, it is perhaps impossible to say.

As the result of our consultation, it was determined to aspirate the tumour. This was accordingly done, and two pints of bloody pus were withdrawn. The portion of the tumour which was situated to the left of the middle line, was dissipated by the aspiration; but the

* With the exception of some slight verbal alterations, the paper is published exactly as it was read.

portion to the right of the middle line remained as well defined as before. As this portion of the tumour appeared at one point to yield an indistinct feeling of fluctuation, it also was aspirated; but nothing was withdrawn.

After the operation, the patient appeared for some days to get relief; and the tar-like discharge from the vagina ceased.

On December 13th, when I next saw the patient, she expressed herself as being much easier; the pain in the abdomen had, in fact, almost disappeared. She was now passing a considerable quantity of pus *per rectum*. The firm portion of the tumour, situated on the right of the middle line, had settled down considerably lower into the pelvis; and a localised swelling, which gave a highly tympanitic note on percussion, now occupied the position of the fluctuating portion of the tumour which had been aspirated. It was obvious that a communication had been formed between the intestine and the pus-containing cavity.

In the beginning of January 1875, the patient was removed to the admirable new hospital, which had recently been erected by the Guardians of the Tynemouth Union. The markedly beneficial and tonic effect of plenty of fresh air and sunlight on this patient, was very remarkable. She rapidly gained colour and flesh, and was able to eat with avidity and enjoyment.

On February 11th, she passed, per rectum, the lower half of a fœtus, which, judging from its length, must, at the time of its death, have been about five months old. The soft parts were macerated and disintegrated, the bones in places being bare and clean. (The specimen, which is in my museum, is represented in fig. 83.)

The firm portion of the tumour, which was formerly situated to the right of the middle line, was now felt to be smaller; its position, too, was altered; it had gradually made its way to the left of the middle line, and finally lay in the left iliac fossa.

After the expulsion of this portion of the fœtus, the purulent discharge from the rectum still continued, and the general condition of the patient improved so much, that she was able to get up and walk about. There was, however, still marked tenderness on pressure over the remains of the tumour; and any movement, such as walking, was attended with pain and uneasiness.

At the end of March, the condition of matters was practically unchanged; and as the patient was most anxious to get out of hospital, and to have the remainder of the fœtus removed, we decided to try what could be done.

On April 4th, the patient having been placed under chloroform, Mr Page introduced his hand into the rectum. An opening the size of a five shilling piece was felt high up in the wall of the gut; the edges of the orifice were firm and well defined. Mr Page succeeded in introducing the tips of two fingers, through this opening, into a cavity, in which the bones of a fœtal head could be felt. Two small portions of bone were removed; but it was found to be impossible, without using an amount of force, which seemed undesirable, to remove the remainder of the fœtus.

The manipulation was followed by some local peritonitis and febrile disturbance; while the portion of the fœtus which remained, again changed its position, passing from the left to the right iliac region.

In the course of a few weeks, the general condition of the patient was so much improved, that she was, at her own request, discharged from hospital.

I heard of her from time to time, through my friend the late Dr Reid of Newbiggin. Soon after her discharge from hospital, she was able to resume her occupation as a hawker, and to tramp from place to place amongst the pit villages of South Northumberland. From time to time fœtal bones were discharged per rectum. Some of these bones, which Dr Reid kindly sent me, I have still in my possession. When I last heard from Dr Reid, the discharge of pus from the rectum had entirely ceased, and the patient appeared to be quite well.

Such are the facts of this very interesting case. The question still remains, and I fear must continue to remain unanswered, was this woman pregnant at the time when she received the injury, or did she become pregnant after the

Fig. 83.—Lower half of a fœtus expelled from the rectum, in a case of extra-uterine gestation.

The tissues are macerated, and the bones are, in places, quite bare and clean. The letter *a* points to the last lumbar vertebra; *b*, to the right iliac bone; *c*, to the right femur; and *d*, to the right tibia.

receipt of the injury. If the patient's statement is to be relied upon, conception must have already taken place at the time of the injury; for she stated most positively, that she had had no connection with her husband—and presumably she had not had connection with any other man (though I have no definite statement in my notes to this effect)—after the receipt of the injury.

Further, it seems impossible to come to any definite and positive conclusion, as to whether the pregnancy was from the first extra-uterine or not.

It is, I presume, within the bounds of possibility, that being pregnant at the time of the assault, the uterus was ruptured by the injury (kick on the lower part of the abdomen) which she received. But while such an occurrence may possibly have taken place, it appears to me to be very unlikely. A much more likely explanation seems to me to be, that which supposes that the fœtation was abnormal, probably Fallopian, from the first.

The fact that the patient had been married for seven years and had not conceived is suggestive, when taken in conjunction with the fact that the pregnancy which developed, or, at all events, was noticed after the injury, was extra-uterine, that some local peculiarity or structural condition existed which prevented normal conception. The fact that the patient menstruated naturally a week before the injury, when taken in conjunction with the subsequent history of the case, seems strongly opposed to the view that the fœtus was ever contained in the cavity of a normal uterus.

To me it appears to be difficult to advance a perfectly clear and satisfactory theory capable of reconciling all the facts of the case. The most probable view to my mind, is that which supposes that the patient became pregnant immediately before the receipt of the injury; that the pregnancy was abnormal from the first—probably intra-mural or Fallopian. My knowledge on these matters is not sufficiently great to enable me to say whether the facts of the case are compatible with the supposition that conception occurred in a bicornual uterus; but so far as I understand the subject, I should think that such a view is much less likely than that which supposed that the fœtation was ectopic.

II.—Case of Extra-uterine Fœtation going on to the Full Time; Removal of a Dead Fœtus by operation; Death from Septicæmia and Hæmorrhage.

The following appears to be an example of the form of ectopic gestation in which the gestation is originally Fallopian, and in which, as Berry Hart and Lawson Tait have shown, the Fallopian cyst ruptures into the folds of the broad ligament, the fœtus continuing to grow until the full time in a sac which, from first to last, is extra-peritoneal.

The notes of the case are briefly as follows:—

M. B., aged thirty-four, was admitted to the Newcastle-on-Tyne Infirmary under my care on the 8th of April 1875, on the recommendation my friend Dr G. T. Beatson.

She had been married for eleven years, but this was her first pregnancy. Dr Beatson believed that the fœtation was extra-uterine. She had never had a miscarriage.

The pregnancy had advanced as far as the eighth month. The abdomen was equally distended by a solid tumour, which extended for three inches above the umbilicus. At the level of the umbilicus the abdomen measured thirty-eight inches in circumference.

A dark brown areola surrounded each nipple; the breasts contained milk; a brown line extended from the pubes to the zyphoid cartilage, the discolouration being most marked between the umbilicus and the pubes.

The outlines of a fœtus could be distinctly felt within the tumour which filled the lower part of the abdomen. The different parts of the child could be manipulated with unusual facility.

The sounds of the fœtal heart were very distinctly heard at a point two inches below the umbilicus, and slightly to the right of the

middle line; the placental souffle was also well heard at a point a little below that at which the fœtal heart-sounds were audible. The patient stated that she occasionally felt the child make slight movements.

Since the earlier months of pregnancy there had been more or less pain and tenderness on pressure over the lower part of the abdomen. Of late, the attacks of pain had been more frequent and the tenderness had become more marked. At the time of her admission to the Infirmary there was great pain all over the position of the tumour; even gentle manipulation of any part of it caused complaints of suffering.

There did not appear to be any fluid in the cavity of the peritoneum.

The radial pulse was 80, the temperature normal, the tongue slightly furred; the patient was very thirsty; and during the four days that she was kept in hospital for observation she vomited occasionally.

On vaginal examination, the os could just be reached with the finger; it *seemed to be very small.* (This statement is underlined in my case book.) I find no statement as to the presence or absence of menstruation in the notes.

The feet and legs were œdematous; and the urine contained a considerable quantity of albumen, but no casts. She complained of tenderness on pressure over the swollen lower extremities, which pitted on pressure and were not red. In short, no local cause for the tenderness was apparent.

The heart, lungs, and other organs appeared to be normal.

Diagnosis.—I felt considerable difficulty in accepting Dr Beatson's diagnosis. He based his opinion that the pregnancy was extra-uterine on the following combination of facts:—The circumstance that the woman had been married for eleven years but had never been pregnant; the marked peritonitis and history of pain during the pregnancy; the facility with which the different parts of the fœtus could be manipulated—the child seemed to be situated just under the skin of the abdomen—in other words, to be contained in a sac, if there were a sac, which was thinner than the normal uterus at the eighth month of pregnancy; and the very small size of the os uteri.

The subsequent course of the case showed that Dr Beatson's opinion was correct, and that my doubts were not justified.

After being kept for a few days in hospital, for the purpose of observation, the patient was sent home.

About the 8th or 9th of May (I have no record of the exact date) labour pains set in, and I visited the patient in conjunction with her family doctor.

At the beginning of the labour, I satisfied myself that the child was still living. The fœtal heart sounds were then distinctly audible and clearly distinguishable in their rhythm from the arterial pulsations of the mother. I speak with absolute confidence on this point, for while in general practice I had carried on an elaborate and prolonged research on the relationship of the heart sounds to the sex of the fœtus. During the five years that I was in general practice I must have attended at least five hundred midwifery cases. In a large number of these cases, I had made repeated and accurate observations as to the frequency of the fœtal heart sounds in relationship to the diagnosis, before delivery, of the sex of the child. The result of my observations on this point was to arrive at the conclusion, that the rate of the fœtal heart varies, not with the sex, but with the weight and length of the child—a conclusion which was reached altogether independently of, and prior to the publication of, Dr James Cumming's observations on the subject.

In the course of some twelve or fourteen hours, the pains became severe and continuous, but the os did not dilate to any considerable extent; its edges did, however, become softer, and there was a considerable discharge of mucus from the vagina and (?) uterus.

As the labour did not seem to be progressing, it was at length decided to introduce a sound into the uterus with the object of definitely deciding whether the pregnancy was extra-uterine or not.

The introduction of the sound showed that although the uterine cavity was decidedly enlarged, the pregnancy was extra-uterine.

This point having been definitely determined, the patient was advised to place herself in the infirmary under Dr Heath's care, with the object of having the fœtus removed by operative procedure.

The abdomen was opened by Dr Heath some eighteen or twenty hours, if I remember right, after the commencement of labour, and a dead, but full-term fœtus, was removed from a thin-walled sac which appeared to be situated in the cavity of the abdomen. The fœtus was not only dead, but it looked as if it had been dead for a considerable time; its skin was peeling. I emphasise this point, for I feel absolutely convinced in my own mind that it was living at the beginning of labour — some eighteen or twenty hours, if I remember right, before it was extracted from the cavity of the abdomen.

The placenta was left *in situ*, and a drainage tube introduced into the cavity of the cyst.

In the course of a few days symptoms of septicæmia developed, and the patient died, partly from septicæmia and partly from hæmorrhage.

On examining the parts after death, I was unable to satisfy myself as to the exact character of the pregnancy; the parts were all so matted together by adhesions that their exact relationship could not be definitely determined. The uterus was enlarged, but otherwise perfectly normal. The large sac in which the fœtus and placenta were situated was attached by dense adhesions and connective tissue to the right side of the body of the uterus. The right Fallopian tube could be traced for a certain distance along the wall of the cyst; the right ovary and fimbriated extremity of this Fallopian tube were not found. The left Fallopian tube and ovary were normal.

There can, I think, be little doubt that the sac was extra-peritoneal, and seated in the subperitoneal connective tissue, and that it had gradually separated up the two layers of the broad ligament.

III.—The Plan of Construction of the Motor Mechanism in the Cortex of the Brain.*

It must be remembered, as Dr Hughlings Jackson has pointed out, that the localised motor centres in the cerebral cortex represent, not muscles, but movements. It may at first sight be supposed that this is a distinction without a difference. And, if each muscle represented a given movement, such would undoubtedly be the case. But it must be remembered, *firstly*, that for the production of the great majority of movements, several individual muscles are required; and *secondly*, that the same muscles, which are concerned in the production of one individual movement, are also, as a rule, concerned in the production of several other movements.

Now, if the cell groups in the cortex, which we are in the habit of terming motor centres, represent, as Dr Hughlings Jackson supposes, and as I myself think, movements, and not muscles, it is easy to see that the destruction of any single cell group, which is concerned in the production of any given individual movement, will cause paralysis of that movement, but will not necessarily produce paralysis of the muscles which produce that movement, unless the function of the muscle or muscles concerned in the production of that movement is to produce that individual movement alone.

If the muscles, concerned in the production of any given movement, are also concerned in the production of several other movements, destruction of the cell-group in the cortex, which is concerned in the production of one particular movement, will only produce paralysis of the muscle-group, in so far as that particular movement is concerned.

As a matter of fact, a circumscribed lesion in the cerebral cortex (a lesion which destroys one of the so-called motor centres),

* I had intended publishing this note in the article on *Congenital Hemiplegia due to an injury received at the time of birth*, which appeared in the ninth number of the *Studies*. I was obliged to cut it out for want of space, but as the subject is one of considerable scientific interest, I take this opportunity of bringing it before the notice of my readers.

is found to produce paralysis of certain muscles. There is, however, no contradiction in this; for each individual motor centre, as we are in the habit of terming it, is in reality composed of many different cell-groups, each of which is connected with the production of some individual movement. For the purpose of economy, as regards space, it is obviously a satisfactory arrangement to have all the different cell-groups, representing the many different movements which any given set of muscles (say, the muscles which move the fingers and thumb) produce, arranged in close juxtaposition. For the steady and co-ordinate production of most movements, the combined action of several different muscles and their opponents is usually necessary. The cell-groups in the motor cortex, which represent the different movements of groups of muscles (muscle-groups), which are in the habit of acting in conjunction, must necessarily be situated in close juxtaposition, and are, without doubt, intricately mixed up. The individual motor centres are not sharply circumscribed and defined. The different cell-groups, which represent the different movements of any given set of muscles (muscle-group), must necessarily, to some extent, run into and overlap one another.

The many different cell-groups, representing the many different movements which the same set of muscles carry out, are intricately intermingled in the limited areas of the motor cortex, to which we apply the term motor centres. The destruction and irritation of localised portions of the motor cortex, which the physiologist can produce experimentally, are at the best very crude and coarse. It is impossible, if a limited area of the motor cortex contains many different cell-groups, representing many different movements, to pick out and destroy any individual cell-group of which it is composed. Experimental destruction of any motor centre, however limited it may be, must necessarily destroy more or less completely all the different cell-groups of which that centre is composed. Hence it is that destruction of a particular motor centre in the cerebral cortex, paralyses, more or less completely, all the cell-groups represented in that centre, and therefore more or less completely paralyses the muscles (*i.e.*, the group of muscles concerned in the production of the different movements) represented in the portion of cortex which is destroyed.

Viewed from the practical side of medicine (the clinical side), it is not perhaps a matter of great importance, whether the motor mechanisms in the grey matter (motor area) of the brain represent movements or muscles, since localised destruction of a motor centre must necessarily interfere, more or less completely, in accordance with the completeness of the destruction, with the functional activity of all the individual cell-groups (movement-producing mechanisms), of which it is composed; and must therefore necessarily produce more or less complete paralysis in the muscles which are concerned in the production of those movements. And we know as a matter of fact, that destruction of limited portions of the motor cortex, whether produced experimentally in the lower animal or by disease in man, does produce paralysis of muscles. But viewed from the scientific side, from the point of view of the neurologist, who is trying to understand and unravel the exact plan on which the intricate mechanism of the brain is constructed and the exact manner in which that intricate mechanism works, the distinction is a radical one.

IV.—Herpes Zoster on the Hand.

Dr Coldstream, of Florence, sent me, a few days ago, the following very interesting note on a case of herpes zoster. It is obvious from this and the other cases which have been brought under my notice, since I directed attention to the subject in the third number of these *Studies*, that herpes zoster affects the hand and fingers much more frequently than I, and indeed others who have written on the subject, had supposed.

Dr Coldstream writes as follows:—

"Dear Dr Bramwell,—Having read with much interest your recent clinical remarks on

herpes zoster, I send you a brief note of a case I have been attending here.

"The patient was a lady, single, about fifty years of age, spare, but healthy. She had just arrived from the Lake of Como, where, while out sketching four days previously, she thought she had taken a chill. I found her complaining of acute pain in the right shoulder joint, shooting down the arm into the hand. There was no fever nor rise of pulse.

"A bright herpes zoster eruption was situated on the dorsal aspect of the posterior axillary wall, and extended down the posterior and outer surfaces and the upper arm, evidently following the track of the musculo-spiral nerve. It then coursed down the radial border of the forearm, and appeared *on both palmar and dorsal surfaces of the hand*, covering about two-thirds of both surfaces. It then extended down the sides of the thumb, forefinger, middle finger, and radial side of ring finger, thus corresponding exactly with the usual distribution of the radial nerve.

"I fancy such an extensive distribution must be very rare. I see two correspondents have sent you note of its occurrence on the hand.

"I find herpes zoster occurs here also in waves. I have seen three cases within the last month. In May last, I saw three cases within a fortnight, two of them being on the same day.

"In this case I have mentioned, the pain in the shoulder and hand lingered acutely after the eruption had faded.—Yours sincerely,

"ALEX. R. COLDSTREAM."

V.—CASE OF FACIAL PARALYSIS OF SEVENTY-THREE YEARS' DURATION.

IN the eighth number of these *Studies*, I published a case of facial paralysis of forty-two years' duration, in which the paralysis developed at the age of one year, and was perhaps the result of an acute inflammation of the facial nerve nucleus (poliomyelitis anterior acuta).

During the past few weeks I have had the opportunity of seeing a very similar case. The patient, an old gentleman, aged 74, walked into my consulting room, with the object, as I supposed, of consulting me on account of facial paralysis. But such was not the case; for his complaints had no reference to the facial paralysis, which at once attracted attention, and had been present for more than seventy-three years.

On questioning him with regard to this facial paralysis, which was left-sided and complete, I ascertained that it dated from an attack of convulsions during teething. It occurred when he was eighteen months old. His right foot was affected at the same time; it had been twisted ever since.

It is probable, I think, considering the age at which the disease occurred, the distribution of the paralysis—the left side of the face and the right leg—and the complete character of the paralysis, which could only be the result of a lesion of the facial nerve or its nucleus, that the lesion in this case was an acute inflammation of the grey matter of the facial nerve nucleus—a poliomyelitis anterior acuta.

The lower lid in this case was much everted, and the conjunctiva, both of the lid and ball, was much injected and thickened.

The paralysed cheek was fuller and rounder than the sound one, and bagged considerably about the angle of the mouth. Nothing could be more striking than the contrast between the appearance of the cheek in this case, and in the case of progressive facial atrophy, which I figured in the eighth number of these *Studies*. In the case of this old gentleman, the cheek must have been absolutely destitute of all muscular tissue, and yet it looked much fuller, plumper, and rounder than the cheek on the sound side.

VI.—The Importance of Sleeping in an Airy and Well Ventilated Bedroom.

Fisherman; aged 26; living in Shetland.

This patient came to the Clinic, complaining of cough, spit, and shortness of breath on exertion. He had spat blood, and the physical examination demonstrated the presence of phthisis at the apex of the left lung. His appetite and digestion were good; he was neither gaining nor losing weight; he did not get hot, and did not sweat at night; the expectoration, though thick and yellow, was only moderate in amount; the pulse frequency was normal. So far as he knew, he did not inherit any tendency to the disease.

Dr B. (to the Students). This is clearly a chronic case of phthisis. The prognosis, as regards duration at all events, seems favourable. The facts that the patient is not losing weight, and that his appetite and digestion are good; that the pulse frequency is not increased; and that there is no febrile rise and no sweating at night, are very favourable indications. There does not seem to be any marked tendency to hæmoptysis in this case. That is also a favourable indication. It is not uncommon to have repeated large bleedings from the lung in cases of chronic phthisis. Aneurisms on terminal branches of the pulmonary artery are by no means uncommon in cases of fibroid phthisis. The rupture of an aneurism on a terminal branch of the pulmonary artery, is the most common cause of copious and fatal hæmoptysis in phthisis. The rupture is by no means always fatal. The ruptured orifice may become plugged with clot, and the bleeding may in this way become arrested. Another danger in chronic phthisis, is the production of waxy (amyloid) degeneration in the liver, spleen, kidney, intestine, or other organs. Cases of chronic phthisis or bronchiectasis, with profuse purulent expectoration, are, in my experience, the most common cause of amyloid degeneration, at the present day. Five-and-twenty years ago, in my student days, amyloid degeneration was very much more common than it is now. Since the introduction of antiseptics, amyloid degeneration has become very much less common than it used to be. Diseased hip joints and diseased bones are not now allowed to suppurate as they used to do. The abolition of chronic and long continued suppuration has prevented a great deal of amyloid degeneration.

It may seem strange to you that a man, who follows the occupation of a fisherman, and who does not appear to inherit any tendency to phthisis, should be affected with the disease. One would hardly expect that a man who breathes the pure air of the ocean during a large part of the twenty-four hours, should be the subject of phthisis. It must, however, be remembered that the sanitary condition of the houses in which people live, who breathe the purest air during the day, is often very unsatisfactory. Phthisis is common in many parts of the Highlands of Scotland. In many parts of the Highlands, people shut themselves up during the night in a box-bed, and breathe the impurest of atmospheres. The benefit which they derive from breathing the splendid fresh air of the Highland hills is not sufficient to counterbalance the injurious effects of their insanitary home-surroundings, and of the impure air which they breathe during the night.

Dr B. (to the Patient). Is your house healthy?

Patient. Yes; pretty well.

Dr B. Is it a good house?

Patient. Oh, yes.

Dr B. Is it damp?

Patient. A little.

Dr B. What sort of a bed do you sleep in? Is it a box?

Patient. Yes.

Dr B. Do you shut yourself up in it?

Patient. Well, not entirely.

Dr B. Do you sleep by yourself?

Patient. Yes.

Dr B. What is the size of the box-bed?

Patient. Six feet by three and a half.

Dr B. Can you turn yourself in it?

Patient. Oh, yes.

Dr B. Is there any ventilation into it?

Patient. There is a small window.

Dr B. Is it open at night?

Patient. No.

Dr B. I suppose you sometimes shut yourself up in the box?

Patient. Yes.

Dr B. (*to the Students*). It is obvious that this patient's surroundings during the night are most unsatisfactory. Nothing could be worse than to sleep in a box-bed in a damp house. It is essential in the treatment of phthisis to see that the patient breathes a pure atmosphere during the night as well as during the day. Badly ventilated, small bedrooms are a fertile source of disease. There is nothing more conducive to good health than plenty of fresh air during the night as well as during the day. The ventilation of a bedroom may appear a very commonplace matter—far too trivial a matter to be mentioned in these *Studies*. I entirely, however, dissent from such a view. I regard it as a very important matter. Most people are far too frightened of fresh air. I strongly disapprove of draughts, but I as strongly approve of an abundance of fresh air. Both for the preservation of health and for the treatment of disease, it is difficult, I think, to over estimate the importance of sleeping in a large, airy, and well ventilated bedroom.

VII.—The value to the Patient of the Aura in Epilepsy.

Male; aged 20; a sailor.

Dr B. What do you complain of?

Patient. A pain at the heart.

Dr B. How long have you had it?

Patient. About eighteen months.

Dr B. Is it always there?

Patient. No, only at times.

Dr B. When do you feel it?

Patient. It comes on at any time.

Dr B. Does anything in particular bring it on?

Patient. If I get excited it comes on.

Dr B. Is it a severe pain?

Patient. Yes; it makes me tremble.

Dr B. Is the pain confined to the region of the heart? Does it extend in any direction?

Patient. No; when the pain comes on I feel something go up towards my heart, and my eyes get dim.

Dr B. Does the pain go up to your head? Does it go up to your shoulders or down your arms?

Patient. No; I feel a swimming go up to the head, and my eyes get dim, and I tremble.

Dr B. Do you ever become unconscious?

Patient. Yes, after the swimming feeling goes up to my head.

Dr B. Have you ever tumbled?

Patient. Yes; I fell off the rigging twelve feet.

Dr B. Did you hurt yourself?

Patient. My leg was sore for eight days.

Dr B. How long have you been taking these turns?

Patient. Eighteen months.

Dr B. How often do you take the turns?

Patient. Often I have had four in a day.

Dr B. Do you ever take them during the night?

Patient. Yes; often.

Dr B. Do you ever bite your tongue in an attack?

Patient. No.

Dr B. Has any one ever seen you in an attack?

Patient's sister (who accompanied him). Yes, he had one to-day as he was coming up to the hospital.

Dr B. What was it like?

Patient's sister. He had to hold on to the railings and he swayed about.

Dr B. Did he appear to be insensible?

Patient. No, it was a slight attack.

Dr B. (*to Patient's sister*). Did you notice if his face twitched?

Patient's sister. No; he was red in the face.

Dr B. Were his eyes twisted?

Patient's sister. Yes.

Dr B. (*to Patient's sister*). Is he ever convulsed in the attacks?

Patient's sister. Yes.

Dr B. Have any of your brothers or sisters, or relations, had attacks like these?

Patient. No; one of my sisters has heart disease.

Dr B. Are you well otherwise.

Patient. Yes.

Dr B. Are you short of breath?

Patient. Yes.

Dr B. Have you ever had any ailment except this?

Patient. I had typhoid fever.

Dr B. Have you ever had rheumatic pains in your joints?

Patient. No.

Dr B. Have you ever had scarlet fever?

Patient. No.

Dr B. Please go into the next room and take off your shirt, and we will examine your heart.

Dr. B (to the Students). The case is evidently one of epilepsy. The fact that the patient says he is short of breath, and that he complains of pain in the region of the heart, suggest the possibility of some cardiac lesion, but there is nothing in his appearance indicative of pronounced cardiac disease. In all cases of epilepsy, it is important to examine the heart. It is by no means uncommon to find mitral disease combined with epilepsy. In some cases of that kind, it is very difficult to say how far the epilepsy is due to, or influenced by, the mitral lesion; but it is certain that, in many cases in which epilepsy and mitral disease are associated, the administration of cardiac tonics, such as digitalis or strophanthus, is a valuable means of treatment. The administration of digitalis, along with bromide of potassium, is in some cases of this kind more beneficial than the administration of bromide of potassium alone.

The patient here returned to the Clinic. After examining the heart, Dr Bramwell stated that he was unable to detect any evidence of organic disease. The urine was also found to be normal.

Dr B. (to the Students). I fail to detect any cardiac lesion likely to produce either pain in the region of the heart or shortness of breath. The mere fact that a patient complains of pain or uneasiness in the region of the heart is not, of course, indicative of a cardiac lesion. In fact, quite the contrary—uneasy sensations, and pain in the region of the heart, or palpitation, especially in young or emotional and excitable people, are *per se* suggestive of functional disturbance rather than of organic disease. If a patient complains only of subjective sensations in the region of the heart, and if there are no evidences of embarrassment of the circulation, especially if there is no shortness of breath, the probability is that the uneasy sensations in the region of the heart are the result of functional disturbance, and not of organic disease. In making a diagnosis, however, we do not rely upon probabilities. The presence or absence of organic cardiac disease can only be determined by physical examination. In the great majority of cases in which a patient complains of pain or uneasy sensations in the region of the heart, or of palpitation, and in which you fail to find, on making a careful physical examination, any evidence of organic cardiac disease, you are justified in concluding that the cardiac symptoms are merely functional. In some cases, however, it is impossible to be absolutely certain. In some cases of true angina pectoris, for example, it is impossible to detect any definite cardiac lesion. Some of the cardiac lesions with which angina pectoris is apt to be associated, such as disease of the coronary arteries, fatty degeneration of the heart, fibroid degeneration of the heart, and small aneurisms at the root of the aorta, are difficult or impossible to detect by physical examination. Fortunately these lesions do not occur, or very seldom indeed occur, in young subjects. The age of the patient is therefore in such cases a very valuable factor in the diagnosis.

My present purpose is not, however, to consider the differential diagnosis of organic and functional cardiac disease. Though this patient complains of pain in the region of the heart, and says he is short of breath, there is no reason, so far as I see, to suppose that he is suffering from any organic cardiac lesion. The fact that mental excitement or agitation pro-

duces the pain in the region of the heart is corroborative of that opinion.

The condition from which the patient is suffering, is epilepsy. He distinctly states that, in some of the attacks, he is unconscious, and his sister says, that he is sometimes convulsed. On one occasion he fell from the rigging, some twelve feet. There can be no doubt, from these facts, that the attacks are epileptic seizures.

In this case, the epileptic fits are preceded by a well marked aura; he feels, he says, a pain in the region of the heart; this is followed by a swimming sensation which mounts up to the head; his eyes become dim; and he then, in the severe attacks at all events, becomes unconscious.

Now, the fact that an epileptic fit is preceded by an aura or warning sensation, is a very important one for the patient. Although the aura is usually of brief duration only, it usually lasts a sufficiently long time to enable the patient to place himself in a position of safety. Epileptic patients should be told to lay themselves flat down on the ground whenever they feel the aura commencing. If they have time, they should loose anything tight which is round the neck. Some epileptic patients are unable to lie down or do anything during the aura. Some patients will tell you that although they know a fit is coming on, they feel fixed, and unable to lie down. Many epileptic patients, who have no aura, fall suddenly down unconscious, and injure themselves severely. They may fall into the fire, or the water, or may crack their heads against the pavement. The aura then is a very important fact for the patient. The same warning sensation usually precedes the fit in any given case of epilepsy. This is not, however, invariable. We sometimes find that the character of the aura changes from time to time. As a rule, however, the fits in any given case of epilepsy are always preceded by the same kind of aura. The aura may consist of any form of sensation which we are capable of experiencing. In some cases, the warning sensation, or aura, is a painful sensation, such as a stinging sensation in the tip of the tongue or in the fore finger. In others, it consists of a flash of light, of a noise, bad smell or taste. Special sense sensations are, however, comparatively rare. In many cases, the aura consists of a peculiar feeling, which patients often find it difficult to describe exactly, in the region of the stomach. In other cases, the aura consists of a creepy or cold feeling in some part of the body. In some cases, the aura is motor, and not sensory. The fit may be preceded by palpitation or fluttering of the heart; in some cases the attack is preceded by localised convulsive twitchings, similar to the localised spasms which occur in Jacksonian epilepsy. In rare cases, an "intellectual" aura occurs; Dr Hughlings Jackson has directed special attention to the "dreamy states" which sometimes precede an epileptic fit. I have met with several cases in which the speech centre appeared to be the first part discharged in an epileptic fit, and in which the aura consisted in the utterance of certain definite words, which were always the same in the same case. I might enumerate many other forms of aura, but I have said enough to show you that the character of the aura is very variable. In fact, the aura may consist of any sensation or motor phenomenon, which results from the discharge of a localised area of the brain cortex; and since all the sensations which we can experience, all the motor actions which we can voluntarily produce, and probably, too, all the involuntary and visceral actions which occur in the human individual, are represented or re-represented in the cerebral cortex, the epileptic aura *may* consist of any sensory or motor or vasomotor phenomenon which can occur in the living body.

It is probable that the aura represents a localised discharge of grey matter in the brain. This is obviously a point of great importance, for, if the theory is correct, the character of the aura may give us a clue to the position of the grey matter which is first discharged in the fit; in some cases—where, for example, there is any question of trephining—this information is of real practical importance.

The practical point, however, which I wish

more especially to emphasise in connection with the aura is this, that epileptic patients whose fits are preceded by an aura should be told to lie down, and place themselves in a position of safety, the moment they perceive the warning sensation.

Some two years ago we had a very instructive illustration of the necessity of the patient immediately obeying the warning sensation. About three years ago a sailor came to the clinic, complaining of epileptic fits. The attacks were preceded by a well-marked aura; the necessity of obeying the aura immediately it occurred was pointed out to the patient; and some bromide of potassium was prescribed. I saw no more of the case for a year, when (his ship having again come to Leith) he again presented himself. He stated that he was very much better, but he further said, that he had been very nearly drowned in one of the attacks. It appeared that on one occasion, while he was mooring his boat to a quay side, he felt the aura commencing. Knowing that the warning sensation lasted for a little time before the fit occurred, he thought he would, before laying himself down, have time to complete the mooring of his boat, He was, however, too late. Before he had completed the mooring, he became unconscious, fell over the quay side, and was nearly drowned. You may be quite sure that *he* will never again neglect the warning. The case is a striking one, and may serve to enable you to realise how important it is to impress upon every epileptic patient the necessity of obeying the warning sensation immediately it occurs.

VIII.—Case of General Paralysis cured by Antisyphilitic Treatment.*

The following are the detailed notes of the case of general paralysis of the insane cured by antisyphilitic treatment, to which I referred in the eleventh number of these *Studies*. The case is so important that I make no apology for reprinting the notes in full:—

Mr A., aged 32, an engineer's draughtsman, consulted me by the advice of Dr Tennent of Glasgow on May 1st, 1882. With the patient Dr Tennent sent me the following statement:—

"Mr A., whom I advised to see you when in Edinburgh, consulted me first in June last on account of dyspepsia. He then recovered. In the beginning of the year he again consulted me, stating that his memory was becoming defective, and that he found considerable difficulty in getting through his work. He had also occasionally felt a tendency to come down on his knees. There was some hesitation of speech; the pupils seemed equal, but I did not subject them to the light test; the patellar tendon reflex was certainly diminished; equilibriation tests were satisfactory. He was evidently extremely nervous about himself, and to avoid the worry arising from business duty, I advised a complete holiday. When he returned, recently, it was evident that though the general health was improved, the condition otherwise was aggravated. Tendon reflex was still more diminished; there was inequality of the pupils, the left being dilated, they were both, however, sensitive to light. Equilibriation with eyes shut was still satisfactory, but he showed some uncertainly in going up and down stairs, and he had evidently experienced considerable difficulty when walking over rough ground. There was however, another feature of his condition which added to my anxiety, viz., he showed a marked tendency to fits of excitement and depression. He displayed very little nervousness regarding his condition, but talked in an excited, happy way of occurrences which had taken place years ago, as well as of the incidents of his holiday. At times he seemed tending to incoherence, and to myself he made several remarks almost of the nature of hallucinations. On the last two occasions I saw him he would, however, hardly give me a reply remaining sullenly silent; he finally stated that he had been startled during the night by people through the wall. His cousin will give you fuller details of his ways at home. There was

* The case was published in *The Edinburgh Medical Journal* (January 1888, page 630).

I understand, a love difficulty some time ago, since which he has been depressed. There is a history of syphilis."

On examination, I found the patient was stout, muscular, and well-nourished. His facial expression was markedly blank, and stupid looking. The right pupil was smaller than the left, but both reacted to light. Speech was very thick, indistinct, and blurred, labials being pronounced with special difficulty. His friends stated that he naturally had a burr, and that his speech was always somewhat thick, but that it had got most markedly worse since his illness commenced. They also stated that when he was staying at Moffat in February, he had had a sudden attack of loss of power and speechlessness; after a little time, he recovered, but was excited for several hours afterwards. Spasmodic twitchings, which were every now and again noticed in the muscles of the face, when the parts were at rest, were very marked, both in the lips and face, on movement or speaking. The facial twitching and speech difficulties were most strikingly displayed when the patient attempted to repeat such a sentence as "papa's performing pony." Fine tremors were also noticed in the tongue when it was protruded. The mental deterioration was marked. At times the patient was depressed, at others happy or excited. Every alternate day he seemed to be depressed and taciturn. He had numerous delusions. On the first occasion on which I saw him, he sat sullenly silent, and it was with difficulty that he could be induced to answer questions. On that occasion, he stated that men were continually watching and following him; that at night he could hear them walking up and down outside the house talking about him.

On the next visit, he was much more lively, and talked freely, first on one subject and then on another; on this occasion, he laughed immoderately, and sometimes without any apparent cause, and stated that he had taken out a patent for a new hair pomade, and that he was going to make a large fortune. On another occasion, he stated that he had seen a large serpent, several yards long, going about the house, and that it was now in his inside; that the men who were watching him were acting upon him with galvanic batteries. His cousin stated that on one occasion he had walked about the house stark naked, and had exhibited no sense of shame, when seen and expostulated with. On several occasions, he would not allow that there was anything the matter with him, seemed very happy and pleased with himself, and talked of his patent pomade and the large fortune he was to make by it. His friends stated that his sleep was very uncertain; some nights he hardly slept at all, but would be constantly getting up and wanting to walk about. As a rule he was, even when excited, easily quieted.

His movements were distinctly unsteady, but there was no distinct ataxia; he was able to stand with his eyes closed and his feet close together. The knee-jerk was absent in both legs. No syphilitic manifestations of any kind were detected in any part of the body. The optic discs were natural; there had been neither headache, vomiting, vertigo, spasms, nor localised paralysis. The bowels were costive, the tongue slightly furred, but in other respects the general health seemed good.

As the result of my examination, I came to the conclusion that the case was one of general paralysis of the insane; wrote Dr Tennent to that effect; and suggested that Dr Clouston should be asked to see the case with me, and to consider the advisability of asylum treatment.

Dr Tennent, in reply, expressed his entire agreement with this opinion, and stated that the specific treatment (iodide of potassium and mercury) which the patient had been taking for some time, had hardly as yet had time to show its full influence.

Shortly after this, about the beginning of May (I have not the exact date), Dr Clouston saw the patient with me. He was of opinion that the case was probably one of general paralysis of the insane, but that there was a possibility of the condition being due to syphilitic brain mischief. He advised that the antisyphilitic treatment should be steadily continued, that the dose of iodide should be increased from 20 to 30 grains three times daily, and that the

head should be blistered. He expressed the opinion that, as yet, there was no necessity to send the patient to an asylum, but advised that he should be kept at home under the charge of an attendant.

This was accordingly done. At the beginning of June, the dose of iodide was still further increased to 30 grains five times daily, the former dose of perchloride of mercury ($\frac{1}{24}$th grain) being continued. The blistering of the head was repeated five times.

On the 12th of June, the patient, who had continued much *in statu quo*, went to North Berwick.

On getting out of bed on the 15th of June, he felt the left side (arm and leg) weak; the weakness gradually increased, and in the course of a few hours amounted to total paralysis (left hemiplegia). There was also, for a short time, inability to speak (temporary aphasia). The attendant stated that during this attack the skin felt hot, and the pulse was quick.

On the evening of the 16th of June, I saw the patient with Dr M'Bain; the febrile disturbance had then completely disappeared, and the paralysis was very much diminished; the patient had, in fact, been able to take a short walk out of doors in the middle of the day. In the course of a few days, the paralysis entirely disappeared. After this attack, the mental condition seemed to undergo some improvement.

On 14th July, there was another sudden attack of right-sided hemiplegia and aphasia. In four days, the paralysis of the arm and leg had again passed off; the aphasic condition continued for some days later; for the first few days of this attack, the urine had to be drawn off with the catheter. The attendant was positive that neither of these attacks of hemiplegia were preceded by any convulsive twitchings.

On 19th July, there was another slight attack of temporary aphasia, without limb paralysis. I have a note on that day to the effect, "that for three weeks there have been no delusions, but that for a week or more the patient has been extremely stupid and apathetic, sitting silent for hours together, and appearing to his attendant to be quite confused in the head." The appetite has been very fair; the bowels, which at the beginning of June were, the attendant stated, very obstinately constipated, have been acting freely. The iodide and mercury have been steadily continued.

On 21st July, the patient returned to Edinburgh; on the 22nd July, he came down to see me at my own house. He appeared to be walking somewhat better, but to be still very stupid and dull. His attendant stated that he was sleeping better. There was still much facial and lip tremor. After the last congestive attack, he had, for one day, complained of his head, and has occasionally since, in answer to a direct question, said that it was sore. It must, however, be particularly noted, that this was the only occasion throughout the whole course of the illness, on which he complained of headache.

On 25th July, Dr Clouston again met me in consultation. We found the patient much *in statu quo;* the vigorous antisyphilitic treatment had apparently produced very little effect; the patient had, it is true, lost his delusions, but the dementia was more marked, the tremor of the lips and face, the affection of speech, and the motor weakness were quite as great, in fact apparently greater. Dr Clouston was now definitely of opinion, that the case was one of ordinary general paralysis of the insane, and not of cerebral syphilis—a view which he recorded some months later in his admirable clinical lectures.

On the evening of 1st August, after being out for a walk, the patient was seized with a rigor. He was immediately put to bed, and in the course of a short time fell asleep. The next morning he was found speechless and paralysed on the right side of the body; at this time he appeared to be quite conscious. In the course of a few hours, he gradually became completely comatose, and he remained so for thirty-six hours. He then became partly conscious, at times wandering, at others highly nervous and excited; on two successive nights, he had to be restrained by force; he was not actually violent, but much excited and apparently frightened, wanting to get up, leave his bed, and go out of the room.

(*To be continued.*)

Studies in Clinical Medicine.

FRIDAY, DECEMBER 27, 1889.

CONTENTS.

I.—CASE OF GENERAL PARALYSIS OF THE INSANE CURED BY ANTISYPHILITIC TREATMENT.

(*Continued from page* 232.)

During this comatose attack, a large bedsore formed over the middle of the sacrum. Some few days after he regained consciousness, a deep abscess burst while he was straining at stool, and a large quantity of pus and blood escaped externally. The attendant immediately plugged the wound, and sent for Dr Dunsmure, who had kindly taken charge of the patient during my absence. Dr Dunsmure found that the patient had lost a large quantity of blood, a deeply situated vessel of some size having given way. The coats of the ruptured artery were so brittle, that it was found impossible to secure the bleeding point by ligature. The wound was accordingly plugged.

After this, in the words of his cousin, "Mr A. almost at once recovered his head, he continued to get better day by day until the beginning of October, when he went back to his home in Glasgow."

At the time of his return to Glasgow he was very much improved mentally, but still far from well.

By Dr Tennent's advice, the anti-syphilitic treatment was continued, and the head repeatedly blistered.

On the 18th of December, the patient was so much improved, that the attendant was dismissed.

On the 8th of January, he returned to work; he has continued to improve steadily since that date.

At the end of 1883, he married.

On 15th July 1887, Dr A. Maitland Ramsay, who had, at Dr Tennent's request, acted as his ordinary medical adviser, kindly wrote me as follows:—"Since Mr A. came under my care, I have had very little to note regarding his case, as he has kept so very well. He takes steadily, twice a day, a mixture of perchloride of mercury with iodide and bromide of potassium. He is always at his work as a draughtsman, and some time ago, when through the dull trade he was thrown out of employment, he exhibited the greatest perseverance and activity in obtaining another situation."

The more detailed report which Dr Ramsay kindly sent me, is as follows:—

"Mr A. was first seen by me in March 1885, when he was suffering from a severe attack of nettle-rash. At that time his speech was so thick that it was with difficulty I could make out what he said. His manner was nervous and excited. In a week, he had got completely over the nettle-rash, and since then he has only consulted me specially on account of what he calls 'fainting fits,' which at first came on very

frequently, but now only once in the six weeks. These 'fits' have all along been periodic, the interval between them gradually lengthening. His wife states that he has no warning of the approach of a 'fit;' the attack may come on at any moment after the usual interval has passed. In the attack he suddenly becomes very pale, and his eyes have a staring expression. There are no convulsive movements, but sometimes a slight twitching of the muscles of his face. If a 'fit' comes on at his work, he stops what he is doing and sits motionless. He does not let his square or his pencil drop from his hand. If an attack comes on during a meal, he all at once stops chewing his food and stares straight before him. He himself states that he does not lose consciousness, and that although he knows perfectly well that he is being addressed, yet he feels quite unable to speak. His wife says, however, that she thinks he does lose consciousness, at least at the very onset of the fit, but as the whole attack is over in less than a minute, it is somewhat difficult to say. He has frequently had an attack in his office, and those sitting at the desk along with him did not observe it—only, he says, that they sometimes remarked afterwards that he was looking pale. There is no loss of memory after a 'fit;' after the attack is over, he begins to talk as if nothing had happened. He may have several attacks in the course of the twenty-four hours. They sometimes come on in bed, and his wife states that those attacks, which come on while he is lying in bed, are of longer duration, and apparently more severe than those which occur during the day time. The day after the fit, he is, he says, perfectly well, only for a time he loses the sense of taste; this disturbance of his sense of taste, however, seems not to be so marked now as it used to be.

"His speech is still a little thick, but quite intelligible; he is regularly at his work, is most painstaking and exact in all that he does, and his memory is very good. He reads a great deal, and takes a very lively interest in all that is going on.

"The left pupil is rather larger than the right; both pupils contract freely to the stimulus of light and during the act of accommodation. There is no disturbance in the ocular muscles; and ophthalmoscopic examination reveals no abnormality in the fundus of either eye.

"The knee-jerk is normal, or rather exaggerated on the left side, and nearly absent on the right, but there is not the slightest unsteadiness in his gait, nor any want of the power of equilibration on making him stand with eyes shut and feet close together."

When last seen by me on October 1887, he stated that, with the exception of occasional momentary "fits of abstraction," which are gradually becoming less and less frequent, he feels perfectly well. His memory is, he says, quite good; his drawing is better than it was for years before his illness commenced; and for the past five years, he has had absolutely no symptoms of mental derangement whatever.

Some physical evidences of disease still, however, remain. His speech is much thicker than it was before his illness; his knee-jerk, as tested through the the trousers, seemed absent in both legs; and the attacks of *petit mal*, though steadily diminishing in frequency, still continue.

There is no longer any evidence of motor impairment. I did not on this occasion detect any twitchings or tremors in the tongue, lips, or facial muscles; the pupils were equal, and responded briskly both to light and accommodation. The patient still had a somewhat heavy, stolid expression, which is probably natural to him; his memory and intelligence seemed active, and, so far as I could judge, in every way natural.

His cousin tells me that, for some time after his recovery in January 1883, he made frequent mistakes in writing, missing out letters and words, and using wrong words and expressions. These mistakes have gradually become less frequent, and are now rarely observed.

Up to the present time, December 1889, the patient has, I understand, continued well.

II.—The Value of Cultivating the Habit of Minute Observation; the Value of Practical Work; a Large Clinical Experience and a Practical Acquaintance with Disease absolutely Essential for Successful Medical Authorship; the Preliminary Apprenticeship Question.

Young Woman; age, 18; pupil teacher.

Dr B. What do you complain of?

Patient. Hoarseness.

Dr B. How long have you been ill?

Patient. Since the Spring. I then had an attack of rheumatic fever, and sore throat; the hoarseness has never gone quite away.

Dr B. Was the rheumatic fever a severe attack?

Patient. Yes. I was in bed for a week.

Dr B. Were your joints swollen?

Patient. Yes.

Dr B. And you were feverish?

Patient. Yes.

Dr B. Have you any cough?

Patient. No.

Dr B. Do you feel well enough otherwise?

Patient. Yes.

Dr B. Have you lost flesh?

Patient. No.

Dr B. Have you gained?

Patient. No. I am much about the same?

Dr B. Is your appetite good?

Patient. Yes.

Dr B. Have you been able to go on with your work?

Patient. Yes; but with difficulty.

Dr Bramwell here examined the throat, and stated that there was nothing to be seen to account for the condition.

Dr B. (*to the Students*). The hoarseness does not seem to depend upon any abnormal condition of the throat or fauces. The parts appear to be quite normal. The hoarseness probably depends on some abnormal condition of the larynx. It will be advisable to examine the heart. Possibly the heart was affected during the attack of rheumatic fever.

Dr Bramwell here examined the heart and reported that it was normal.

Dr B. (*to the Patient's mother*). Has she got paler lately?

Patient's mother. Yes, very much.

Dr B. (*to the Students*). It will be necessary to examine the condition of the larynx; the fact that the hoarseness has continued for such a long period of time is suggestive that it depends upon some definite organic cause. Tubercular laryngitis is one of the conditions which might produce it. There do not, however, appear to be any of the other symptoms, which one might expect, if the case were one of tubercular laryngitis. There is no cough; the patient has not lost flesh; her appetite is good. The only abnormal condition which we have detected, in addition to the hoarseness, is anæmia. It is not likely that that is the cause of the hoarseness, but it is well to remember that in some cases of tuberculosis, whether of the lungs or the larynx, the tubercular affection is preceded by, or accompanied by, anæmia.

A laryngoscopic examination, which was made after the clinic, failed to show any definite local alteration in the larynx. The exact cause of the hoarseness was consequently undetermined. Iron and arsenic were prescribed for the anæmia.

Dr B. (*to the Students*). Hoarseness is sometimes associated with disease of the heart, and with disease of the great vessels within the thorax. As you all know, aneurisms or solid intra-thoracic tumours, which press upon or involve the recurrent laryngeal nerve, may produce either spasm or paralysis of the larynx; in some cases of intra-thoracic aneurism and intra-thoracic tumour, very marked laryngeal symptoms, such as hoarseness, loss of voice, a ringing, clangy, metallic cough, or sudden attacks of dyspnœa, are present. When laryngeal symptoms are due to the pressure of thoracic aneurism on the recurrent laryngeal nerve, one can generally detect the presence or absence of spasm or paralysis of the left vocal cord, on examining the larynx with the laryngoscope. In the great majority of cases of this kind, the left vocal cord is alone involved; but in rare instances, pressure on the left recur-

rent nerve seems curiously enough to produce spasm or paralysis of both vocal cords. In all cases of suspected aneurism or solid intra-thoracic tumour, the larynx should be carefully examined. In some cases in which the left vocal cord is paralysed, there are no marked laryngeal symptoms. In cases of suspected aneurism or intra-thoracic tumour, one should not, therefore, be content to ascertain the presence or absence of hoarseness, loss of voice, cough, &c.; it is necessary, even when the voice seems quite normal, and when neither cough nor dyspnœa is complained of, to observe the condition of the left vocal cord by the aid of the laryngoscope, just as it is necessary, in suspected cases of cerebral tumour, to examine the condition of the optic disc with the ophthalmoscope, whatever the condition of vision, *i.e.*, whether vision is good or not.

In most cases in which the left vocal cord is paralysed, you would, of course, expect to have some laryngeal symptoms, but in some cases laryngeal symptoms are entirely absent, or are so slight that they do not attract the attention of the patient. Remember then, in all cases of suspected aneurisms or solid tumour within the thorax, to examine the condition of the left vocal cord with the laryngoscope.

In some cases of pericarditis, there is hoarseness; the alteration of voice may indeed be the only noticeable symptom in pericarditis. In many cases of pericarditis, the onset of the pericardial inflammation is unattended with cardiac pain, or any other symptoms directly suggestive of cardiac disease. The occurrence of hoarseness during an attack of rheumatic fever, or in the course of a case of cirrhosis of the kidney (in which affections pericarditis occurs more frequently than in any other conditions), should suggest a careful examination of the heart. Any symptom, which suggests the onset of a pericarditis, is of great value. I do not, of course, mean to say that the occurrence of hoarseness in a case of rheumatic fever is indicative of pericarditis. Far from it. In the great majority of cases, in which hoarseness occurs in the course of acute rheumatism, it doubtless depends upon other conditions, such, for example, as laryngeal catarrh or ordinary sore throat. Probably the hoarseness in the particular case, which we have just been investigating, was due to one or other or both of these conditions. But when hoarseness does occur in the course of acute rheumatism, it is well to remember, that it *may* possibly be due to inflammation of the pericardium. Although the occurrence of acute rheumatic pericarditis is, in many cases, unattended with any pain in the region of the heart, or any symptoms directly suggestive of cardiac derangement, yet the onset of the pericardial inflammation is not unfrequently attended with distinct alterations in the physiognomy and general appearance of the patient. In many cases, a careful observer is struck with the fact that, with the occurrence of pericarditis, the patient does not look so well. It is obvious, from the facial expression, and whole appearance of the patient, that something has occurred. In some cases of acute rheumatic pericarditis, the onset of the pericardial inflammation is attended with a pale, pinched, and anxious—almost collapsed—expression; in other cases, the face is flushed. The late Dr Sibson suggested that these alterations of the countenance, flushing or pallor, are probably due to reflex disturbance of the vasomotor centre, the pericardial inflammation acting as the source of the peripheral irritation. Be that as it may, it is certain that the only external indication of the onset of a pericardial inflammation is, in some cases of acute rheumatism, the change of physiognomy which I have just attempted to describe. These changes are not, of course, distinctive of pericarditis; they, like hoarseness, are only suggestive. They are, however, of great importance. They show that the patient is not so well. They suggest that something has occurred. What that something is can only be determined by careful investigation and examination. If that something is pericarditis, one would expect to find evidence of the pericardial inflammation (in the form of a to and fro friction murmur, and perhaps of increased pericardial dulness) on making a physical examination of the heart.

I cannot too strongly impress upon you th

necessity of cultivating your powers of observation, and of getting into the habit of noting the most minute details in the physiognomy and general appearance of the patient.

Apparently trifling alterations often give us most valuable information.

In a case of continued fever (typhus or typhoid), you may find, for example, some morning, that a patient, who has been previously lying on his back, listless, and, as it were sunk in the bed, has turned on to his side. That change, to the uninitiated so apparently trifling, is a change of very great significance. On it alone, you may, in most cases, with perfect confidence, hazard a very definite opinion that the patient is better.

Again, in the case of acute croupous pneumonia, the appearance in the sputum of a few black specks, may enable you to predict that resolution is about to occur. In more than one case of acute croupous pneumonia, the occurrence of a few minute black particles in the sputum, has enabled me to predict the speedy onset of resolution, and this, before there was any decline of the temperature or any other evidence of improvement in the general condition of the patient. I remember one case of acute croupous pneumonia, in which I gained great credit by means of this very simple observation. The patient, whom I was attending, now a number of years ago, was in a good position, and his life was a very valuable one; the attack of pneumonia was severe, and for a time it seemed doubtful which way things would turn. One morning, when the attack seemed at its worst, the sputum, which had previously been scanty, tenacious, and rusty—the typical apple-jelly sputum of acute croupous pneumonia—was slightly more copious, and I noticed that it contained some minute black particles. Knowing that this was, in some cases at all events, an indication that resolution was about to occur, and that catarrhal elements were in all probability being expectorated from the previously stuffed-up air cells of the lung, I ventured to predict that a change would probably occur in the course of a few hours, and that the patient would in all probability pull through. Fortunately, both for me and for the patient, the result was as I had expected; by the observation of this apparently trifling point, and by the confident opinion which I felt justified in basing on it, I have reason to know that I gained considerable credit both with the patient and his relatives. Don't let my remarks on this point be misunderstood. I have no intention of saying that the presence of black particles in the sputum of a case of acute croupous pneumonia is indicative of a favourable termination. What I do mean to say is, that in some cases of acute croupous pneumonia, in which the sputum has previously presented the characteristic apple-jelly character, the appearance of black particles in it, is suggestive of commencing resolution; and that, if other things are favourable, the point may be made use of for prognosis. We know that if a patient with acute pneumonia can be enabled to tide over the attack so as to reach the stage of resolution, that in all probability he will rapidly improve, and soon get thoroughly and completely well. I don't mean to suggest that the presence of black particles in the sputum of acute croupous pneumonia is always suggestive of commencing resolution, or that all cases of acute croupous pneumonia, in which black particles appear in the sputum, recover.

I am, however, very anxious to impress upon you the importance of observing every minute detail connected with disease and sickness, and of studying with the closest attention and care every case which comes under your observation, however commonplace it may at first sight appear to be. You must try and see and observe these little points for yourselves. Book-knowledge and book-work are all very well, but, though absolutely necessary and essential, they are altogether inferior to the knowledge which can only be acquired by the observation of disease in the living patient. The only real knowledge of disease is that which is acquired at the bedside by careful and minute observation of the living patient. A real, practical, living knowledge, so to speak, of disease, gained by experience and observation for oneself, is infinitely more valuable than any amount of mere book learning. You may read books till you are

black in the face, but however great your reading, however great your natural ability and cleverness, you will know really little or nothing of disease until you have studied or observed the facts of disease as they occur in the living man.

One sometimes hears people talk of a man's cleverness as a writer of medical books; they seem to think that if a man has acquired, by dint of hard labour and much practice, or, what is probably very rare, has inherited or been born with, the mere power of writing, that he can write medical books. Well, a man who possesses and has acquired the facility of writing, might doubtless write and produce medical books, just as he might write fiction or poetry, but it is absolutely certain that the medical books at all events, which were the result of mere literary skill alone, would be arrant medical rubbish. No one who knew anything about medicine would read them. It may perhaps be possible, by mere innate ability and literary dexterity, to write a work of imagination or fiction, which is worth reading, but it is absolutely impossible for any one, however great his innate and literary ability may be, to write a scientific or medical book, which is worth reading, unless he possesses a real and practical acquaintance with the subject he is writing about. Even for the production of a high class work of fiction, immense study and a first hand knowledge of the subjects touched upon, are essential for success. Between the works of a George Eliot and the ordinary sensational novel, there is a wide gulf fixed; just as there is an almost unbridgeable distance between such a book as Hilton Fagge's Principles and Practice of Medicine and an ordinary medical "pot-boiler."

In science and medicine, a real and practical acquaintance with the subject, in all its bearings, is the only basis for true success. No amount of innate ability, no amount of literary skill, will enable any one to produce a medical book worth reading, unless he possesses this fundamental requirement of success.

One sometimes hears a successful medical author described as a clever writer. A clever writer! The people who talk in that way only show their ignorance. They know nothing about medical writing. You may be sure that they have never written anything worth reading themselves. They are obviously unable to appreciate the most elementary requirements necessary for successful medical authorship. To produce a really good book on practical science or practical medicine, the writer must be practically familiar with his subject, and, as I have already pointed out, a real knowledge of disease can only be acquired by an extensive clinical experience. This is the first essential, the very basis for success. Without this, no one can or ever will produce a work in any department of practical medicine which is worth reading.

The second element for successful medical authorship is, I consider, sound judgment. A man who attempts to write a good systematic treatise on any scientific subject, or on any department of practical medicine, who is not content to ignore or slur over difficult and disputed points, but who conscientiously endeavours to meet every difficulty and to consider every doubtful and disputed point connected with the subject, must be able to weigh evidence correctly, and to test the many different opinions which exist on every medical subject by his sound knowledge and practical experience. Unless a man has a real practical acquaintance with the diseased conditions he is writing about, he is totally unfitted and unable to give a judgment on any difficult or disputed question connected with practical medicine, however logical his mind, and however unprejudiced his opinion may be.

It is only right, in my opinion, that these facts should be prominently made known. Those of us who are doing our best to advance medicine, and to teach others, by recording our experience of disease, have no light task before us. Talk of labouring work! why, labouring work is nothing to the work required for the production of a big book on medicine. To acquire the practical knowledge which is essential for success, requires years of reading, of study, and of patient bedside observation; to produce the

book, once the material—the practical knowledge and experience, and familiarity with the opinions of others—has been gained, requires enthusiasm, an invincible determination to succeed, the most persevering industry, and demands months or even years, of sustained effort and continuous work. When we think of the unfair treatment which the result of all this labour—the outcome of all these years of continuous toil and effort—sometimes (though for the credit of medical journalism it must be stated quite exceptionally), receives at the hands of the critics, one is perhaps for the moment inclined to ask whether the game is worth the candle. The doubt is, however, merely temporary. Thank God, the profession, as a whole, is fair-minded and impartial. Sooner or later every man's work will undoubtedly receive its due. For the moment, the opinion of the critics may deal unfairly with it, and may unduly exaggerate its merits on the one hand, or underestimate its value on the other. But rely upon it, that in the end, every man's work, and which is still more important, every man's true character and real worth, are pretty correctly estimated by the profession. There are doubtless some exceptions to this as to every other general rule, but the exceptions are certainly not numerous.

Those of us who are doing our best to advance medicine, by recording our experience, and by writing medical books, have, I maintain, a right to expect that our writings shall be criticised in a fair and impartial spirit. All we ask for, or rather, I should perhaps say, all that we are entitled to ask for, is a fair field and no favour. We have a right to expect that our writings should be reviewed by competent and fair-minded men, and in a spirit of judicial impartiality. The object of the reviewer should be, not merely to pick out the bad points—though these should most certainly be prominently indicated—but to weigh the merits of the work as a whole. In my opinion, it is the duty of the reviewer to look for the good points quite as much as for the bad points, and to indicate the strong points quite as much as the weak points in any work which he criticises. In short, the criticism should be made in a fair and liberal spirit; the reviewer should always, in my opinion, endeavour to commence his work with a bias, if he must start with a bias at all, in favour of the author. If after a full and careful consideration of the merits of the work as a whole, he conscientiously comes to the conclusion that his criticism ought to be unfavourable, by all means let it be unfavourable. The reviewer of a book has a public duty to perform. If he conscientiously believes that the book is a bad one, it is his duty to point out its defects. The profession has a right to expect that it should be correctly and fully informed of the true merits, whether good or bad, of every work which is reviewed in the medical journals. All that we medical authors have a right to expect is, that our writings should be carefully and fully considered by competent reviewers, and that their criticisms should be just and impartial.*

It seems to be sometimes forgotten that a man who writes a book stakes his reputation on the venture. If the book is a weak one, or a poor one, or a positively bad one, if it contains gross errors, the loss of credit which the author sustains, and quite rightly sustains, is certainly greater than the increased credit and reputation which he gains if the book is a good one. A man who never writes at all runs no such risk. Do not, however, let it be supposed that I have any intention of implying that the men who do write ought to be considered superior, merely because they write, to the men who do not write. Many of our most able men and most reliable practitioners never write at all. What I do, however, wish to point out is this, that a man who does write voluntarily runs

* I must carefully guard myself against having it supposed that the remarks which have just been made on reviewers and reviews are prompted by any personal considerations. The notices of my own books which have appeared, whether in British or foreign journals—with the exception of one journal alone, which has been peculiar in the estimate which it has placed on my writings—have, almost without exception, erred, if they have erred at all, on the side of leniency. Personally I have nothing to complain of. I have only to thank my critics for the generous, and I fear, in many cases, far too favourable estimate which they have placed on my writings.

the risk of adverse criticism; that he voluntarily runs the risk of damaging his reputation as well as of bettering it. The men who never write run no such risks, and it is perfectly certain that if some men, who have attained to professional eminence and great practice, had written, that their eminence and reputation would have been proved to be fictitious and undeserved. Just as it is perfectly certain that some medical authors have, for the moment at least, gained infinitely more credit and reputation, by their writings, than the real merit of their work in any way entitled them to.

What I desire in particular to emphasise is the fact, that mere cleverness, mere ability, mere literary skill, will never enable any man to write anything which is really worth reading, anything which is likely to last, on any subject connected with practical medicine. I repeat that, without extensive practical experience, and without a real, first-hand knowledge of disease, no man, whatever his natural ability and literary gifts may be, will ever attain any true eminence as a writer on practical medicine.

The only basis of real and true success in practice, or, as I have attempted to show, in medical authorship, is an extensive clinical experience and a sound knowledge of disease as it exists in the living patient.

The necessity for a more extended practical training for our medical students (I do not refer to the Edinburgh medical students alone, but to all students) is now almost universally recognised. On all hands we have it stated that the young practitioner, on his entrance to practice, is in many instances very inadequately qualified to deal with disease. His book knowledge may, it is said, be extensive, but his practical knowledge of actual disease, even of such common diseases as measles and scarlet fever, is, it is stated, sometimes dangerously and scandalously deficient. The statement is I believe in some instances justified. Here in Edinburgh, where the number of students in proportion to the number of Hospital beds is much greater than elsewhere, the difficulties of supplying the medical student with a sufficient opportunity of studying disease in the living patient is enormously great. If I may judge from my own observation and experience, I should say that the students who are trained at some of the smaller schools of medicine, have at the date of their qualification, in some instances at all events, a greater and wider practical acquaintance with disease, as it occurs in the living individual, than the students who are trained in Edinburgh.

At the start in practice, the student who has been trained in a small school is, I believe, in some instances at an advantage. But it is only at the start. Here in Edinburgh the medical student receives a magnificent scientific and theoretical education. On his first entry into practice, he may be, for a short time perhaps, a little at sea in dealing with the more common diseases and accidents of every day life; but it is only at the start; he enters practice with a broad foundation, and with a splendid theoretical and scientific training. His comparative disadvantage as a practical man, even granting that it exists, and it has I believe been very much exaggerated, soon rights itself, whereas the man who has received an inadequate theoretical and scientific training is never, or is seldom if ever, able to make good his deficiencies. I have no hesitation whatever in saying that, after having been for two or three years in practice, the man who starts with the broad foundation of scientific training and theoretical knowledge is infinitely the abler and sounder practitioner of the two. If I were obliged to be treated by either of the two men, at the time of their first entry upon practice—but certainly if I had my way I would prefer to be treated by neither—I would, I think, be disposed to give the preference to the man with the greater practical knowledge; but if I had to make my choice between the two men, after they had been for two or three years engaged in practice, I have no hesitation whatever in saying that I would select the man who had entered practice with the smaller amount of practical knowledge of disease, but with the broader theoretical knowledge and better scientific training. I speak, of course, generally. I refer to the

average type of man who is turned out by the two different methods of teaching and training. I do not for a moment, of course, mean to suggest that because a man starts with a well trained mind, and with a broad basis of theoretical knowledge, that he will necessarily, after he has been engaged for two or three years in practice, turn out to be the sort of practitioner one would like to be treated by, and that he will necessarily be a sounder, and better, and abler practitioner than the man who starts with a greater practical knowledge of disease, but with a poorer scientific training, and with a narrower basis of theoretical knowledge. There are of course innumerable exceptions to any general statement of this kind. All I mean to say is that, given two or three years of actual experience in dealing with disease in practice, the one system is, in my opinion, more likely to produce a sound and reliable practitioner than the other.

A perfect system should of course combine the advantages of both methods. Our Edinburgh students do not, in my opinion, see enough of actual disease. The time which is allotted to the preliminary and scientific subjects, I mean all the subjects which are included in the first and second professional examinations for the M.B.—as compared with the time which is allotted to the practical subjects (medicine, surgery, midwifery, clinical medicine, clinical surgery, gynæcology, diseases of the eye, ear, throat, and skin, insanity,—and I should include in the final subjects, therapeutics) is proportionately far too great. Or to put the matter more correctly and more justly, the time which is allotted to the final subjects as compared with the preliminary and scientific subjects is far too short. In my opinion, far too short when the extent and importance of the final and practical subjects are considered. I do not mean to imply that too much time is spent on the subjects comprised in the first and second professional examinations. Quite the contrary. The time devoted to these subjects is, in my opinion, by no means too long. I should leave these subjects just as they are at present (transferring therapeutics to the final subjects), but I should extend the time required for the study of the final subjects. It is not enough, in my opinion, to extend the curriculum to five years. It is, I think, very desirable that every student should be obliged to spend two complete years in the study of the final subjects. I should like to see it made compulsory, for every medical student to spend two complete years in the study of the final subjects after he has passed the second professional examination. I refer not merely to the university examinations, but to all the qualifying examinations. It is, I suppose, practically certain, that within a very short time, the period of study will be extended to five years. This is a change which I have long advocated, but unless the student is obliged to spend the additional year in the study of the practical subjects, and more especially in clinical study, the reform will, in my opinion, be inadequate. It is essential, I think, to insist that the additional year be devoted to the practical subjects and to clinical study.

With the object of making the student at the time of his entrance into practice more practical, and of affording him an opportunity of acquiring, during his career as a student, a knowledge of the more common forms of ordinary illness, a suggestion has been made to revive the system of apprenticeship. One proposition is, that before the student commences his course of study at a university or medical school, he should be apprenticed for one or two years to a general practitioner, or attached as a pupil to a dispensary, workhouse infirmary, or public hospital. The other is, that six months or a year should be passed as an apprentice, after the ordinary course of training at a medical school has been completed.

Now, in considering the advisability of reviving the old apprenticeship system, it may, I think, at the outset be conceded, that before a medical student is allowed to engage in practice on his own account, a reasonable guarantee should be demanded by the licensing and qualifying Bodies, that he has a sufficient and satisfactory knowledge of the more common diseases and ailments; that he is at least able to recognise

the more trivial as well as the more serious forms of disease, when he meets with them; that he is sufficiently acquainted with the treatment of these common ailments; and that he is practically familiar with, and has personally practised the more common forms of manipulative procedure, such as setting a fracture, passing a catheter, and the like.

But the question naturally arises, whether the apprenticeship system is the only or the best means of acquiring this necessary information; and if so, whether the period of apprenticeship could be passed before or after the ordinary course of education at a university or medical school.

The subject is so extensive, that I shall not on the present occasion, at all events, attempt to consider it fully. I am, however, anxious to consider the advisability of instituting a preliminary system of apprenticeship. I have no hesitation whatever in saying, that, in my opinion, an apprenticeship, at the beginning of the student's career—before the ordinary course of study at a medical school has been passed—is wrong in principle, and is calculated to have a damaging and disastrous effect on medical education, and upon those students who are obliged to submit to it.

Some of the advocates of the apprenticeship system seem to argue that, if you grant that an apprenticeship is desirable at all, it matters little whether the period of apprenticeship be passed before or after the ordinary course of medical training and hospital study.

In my humble opinion, the two propositions are essentially and radically different in principle. In my judgment, the fundamental principle, as to what is the right method of acquiring knowledge and what is the true method of medical training and education, is involved in the question. The two methods seem to me to be absolutely and diametrically opposed. The question is, I consider, a vital one, and I do most sincerely hope, in the best interests of medicine and the medical practitioners of the future, that the General Medical Council will, when this matter comes up for consideration, determine, once and for ever, and with no uncertain voice, that an apprenticeship at the beginning of the student's career, shall on no account be permitted.

No one denies that if the diagnosis, prognosis, and treatment of disease are to be based on rational principles—and not on mere empiricism—that a knowledge of anatomy, of physiology, and of those pathological processes which are associated with the different morbid conditions, which we term diseases, is absolutely essential.

Now to apprentice a young lad fresh from school to a practitioner or to a hospital with the object of bringing him in contact with disease, and expecting him to acquire a knowledge of the more common and ordinary diseases, and of the methods of dealing with them, and of treating them, before he knows anything whatever of the structure and functions of the human body, when he is absolutely and entirely ignorant of the fundamental facts on which all rational diagnosis, prognosis, and treatment must be based, is, I maintain, to cultivate in him a spirit of pure empiricism.

Such a method of training is, I maintain, calculated to do the student a serious injury. Such a system is, I maintain, totally opposed in principle to everything which is best in modern medicine. Such a system is, in my opinion, calculated to do damage to the student, to teach him wrong methods of diagnosing and treating disease. It is perfectly true, of course, that many students trained in such a method at the commencement of their career, would by their subsequent course of study in a medical school, unlearn these wrong methods; but it is equally certain that many would not. In many instances, the erroneous methods of arriving at conclusions, and of looking at disease, which were acquired during this preliminary apprenticeship, would never, I believe, be entirely forgotten. In far too many instances the wrong method, which was first learned, would probably leave some impression, and would tend to produce an empirical frame of mind through the whole subsequent professional life.

It is claimed, that the great advantage of a preliminary apprenticeship (an apprenticeship *before*

the regular course of study in a medical school) is, that the mind of the student is then more impressionable and plastic, and, therefore, better fitted to receive the routine lessons of ordinary practice, and to gain elementary knowledge of the ordinary and common diseases of every-day life, than at a subsequent period of professional life. To my mind, this argument tells all the other way. Any one who has had much to do with medical education and medical students, must surely be aware, that one of the greatest difficulties with which both the student and the teacher have to contend, is that in many instances the student commences his medical course with a very imperfect general education, and without having acquired the most important faculty which his school training ought to give him, viz., the power of concentrating his attention on the subject in hand, and the knowledge of when and how to work. In my opinion, the amount of knowledge and information which the school boy acquires, important though that is, is by no means the most important object of his school training. Far more important, in my opinion, than the acquiring of any amount of mere fact-knowledge, is the training and disciplining of the mind, and the cultivating in the individual of the power, the habit, the method, and the love of work.* Any system of preliminary education and training, which is likely to impair the habit of regular and systematic work, and which is calculated to encourage wrong and unscientific methods of looking at disease, and of acquiring medical knowledge, and which tends to produce superficiality, to foster purely routine methods of practice, and to produce empiricism, is, in my opinion, to be deprecated.

By all means let us make the medical curriculum as practical as possible, let us remember that our medical students are intended to become practitioners of medicine, and that they are not intended to become microscopists, anatomists, physiologists, and chemists; but at the same time let us take care that in our efforts to make the student more practical, we do not do him and the medicine of the future a serious injury, by putting him in a position in which he is likely to acquire an erroneous method of studying and of treating disease, and in which his habits of diligence and regular study are likely to be loosened and weakened, rather than consolidated and strengthened.

I can conceive, in many instances, nothing more likely to give the student a distaste for the hard and systematic work, which the course of study at a medical school now-a-days demands, nothing more likely to deteriorate his mind and to undo the benefits of his previous school training, nothing more likely to unfit him for deriving the full advantage from his subsequent regular course of medical study, than obliging him to pass a year in preliminary apprenticeship, and in what must necessarily be, in many cases at all events, the observation of disease in a routine and altogether superficial manner.

The medicine of to-day is a very different matter from the medicine of fifty or even of five and twenty years ago. The amount of information which the medical student of to-day is expected to acquire during the brief period of his medical curriculum, and which the medical student of the future will necessarily be expected to acquire, however much the medical examinations of the future may be modified, however much more practical they may be made, is something prodigious, and is continually and rapidly increasing.

I entirely agree with those who think that the medical student of our day is overworked, and that the amount of time which is allotted to practical and clinical work, is, in comparison with the amount of time allotted to scientific and theoretical work, far too short. But to attempt to remedy this state of matters, by making the student pass a preliminary year in an apprenticeship, would, I believe, be a grave error. The remedy, in my opinion, lies in an opposite direction. What we want is, I think, a higher standard of preliminary education, and a longer curriculum, with more time at the end of the curriculum and prior to qualification, devoted to the study of disease in

* I purposely omit any reference to the formation of character and the strengthening his powers of self-control.

hospital and dispensary practice. By raising the standard of preliminary education, not only for men entering the Universities, but for every qualifying Body, we would prevent the badly trained and incompetent men getting into the profession; and by prolonging the period of study, and insisting upon more hospital and clinical work at the end of the curriculum, it would not, I think, be difficult, provided that the final examinations were made more clinical and more thorough, to ensure that, on qualification, our young medical men were capable of dealing with the minor ailments and the more common diseases of everyday life.

It is frequently alleged that the young medical men of to-day make bad assistants, and that they are not only incompetent, and unable to recognise and treat such common diseases as measles, but that they are so puffed up with their own self importance, and with the theoretical knowledge that they possess, that they give themselves airs, and think themselves superior to their principals, that they take badly to the routine of practice, and that, as assistants, they are often more bother than they are worth.

Now, speaking generally, I can honestly say that, so far as my experience enables me to judge, these statements are greatly exaggerated —more especially when applied to the best class of our students. In my opinion, the better the student and the more highly he has been trained, the more enthusiastic is he about practice, and the more eager to avail himself of every opportunity of acquiring practical knowledge by studying and treating disease amongst the very poor, and of performing the duties which fall to the lot of an asssistant in private practice. I believe that the young medical men who are now turned out by our best medical schools, as a rule, make good and capable assistants, and I am strongly inclined to doubt whether the statements, which are from time to time published with regard to the incompetence of assistants, can be applied with any degree of fairness to the average assistant. Doubtless they are true in individual instances. But we hear of the failures and imperfections. Like the successes of the bone-setter, they are trumpeted about. I would ask those who favour a preliminary system of apprenticeship, whether they think that the men who, as newly qualified medical men, are too conceited to act as assistants, would under the system which they propose be likely to make good medical students. For my own part, I am strongly disposed to think that the same disposition of mind which would make a man, on qualification, so puffed up with his own self-conceit as to unfit him for the duties of an assistant, would, if he were obliged to pass a year in a preliminary apprenticeship, also unfit him for satisfactorily performing the duties of a medical student.

After a year of preliminary routine, passed in what I suppose he would term the practical study of disease, without any regular and systematic study, and merely occupied in such trivial duties as a raw lad, without any previous knowledge of medicine, could be expected to perform, he would, I should fancy, take badly to the work which the medical student of to-day is expected to perform. He would be disposed, I should fancy, to pride himself on his great practical knowledge, to neglect the dissecting room, the laboratory, and the class room; he would be apt, I should think, to carry on his study of disease in the wards of the hospital in the same superficial, routine, and unscientific manner in which he had commenced to study it during his apprenticeship.

I would ask those members of the profession who advocate a preliminary apprenticeship, whether they have fully considered the practical side of the question.

The medical practice of to-day, the practitioner of to-day, and the public of to-day are not the same as the medical practice, the practitioner, and the public of fifty years ago.

What duties would they expect the raw lad, absolutely and totally ignorant of everything relating to disease, to undertake and to perform?

I suppose that, in many instances, the first year's apprentice of fifty years ago, was little better than a bottle washer, a mere surgery drudge. These were doubtless the only duties

which, at the commencement of his apprenticeship, he was really able and fitted to perform. In the course of a few months, or perhaps a year, he might perhaps be allowed to try his 'prentice hand at the pulling of a tooth or the bandaging of a leg, possibly at the opening of an abscess, or in the visiting of a case of measles. But it may be safely said that, during the first year of his apprenticeship, he was, in most cases, little better than a surgery boy, and that he gained little even of the routine knowledge of disease. I cannot for my own part see how the raw and inexperienced lads of the present day can, in the average run of cases, be expected to do or to learn much more.

Then again, if every medical student is to be obliged to pass a year as an apprentice, previous to the commencement of the ordinary period of medical study, who are going to take these apprentices?

I suppose it is a moderate computation to say, that at least 1500 or 2000 students commence the study of medicine in this country every year.

How many of the 25,000 medical men on the medical register are, I would ask, prepared to take such apprentices; and of the number that would be willing to take such apprentices, how many, I would ask, are fitted to train and to teach them; how many would be willing and able to devote the time and attention which would be necessary to make the period of apprenticeship really beneficial?

It is perfectly certain, that the apprentice would be of no use whatever to his principal, except as a message boy or a surgery boy. If the apprentice were to derive any benefit whatever from his apprenticeship, his principal would have to act the part of tutor, and to devote a large amount of time and trouble to the training of his pupil. The general practitioner of to-day has, as it is, a hard time of it. If competent men are to be asked to undertake the extra duties involved in the training and teaching of apprentices, they will have to be well paid for their trouble. The cost of the medical curriculum is already sufficiently great; and it is certain that the parents of intending medical students will object to defray the costs of a preliminary apprenticeship, unless they are well assured that they will get good value for their money.

Then, again, do the advocates of the proposed preliminary apprenticeship, suppose that the public will approve of their proposal? Will the public be content to be practised on by young lads, who are absolutely ignorant of the very rudiments of medicine? I do not think so.

To me, a system of preliminary apprenticeship seems both wrong in principle, and unworkable in practice. It may be that I fail to understand the proposal. Although I have expressed myself strongly, I have endeavoured, so far as I do understand the question, to represent it fairly. The matter is, I consider, a vital one, for the welfare of medical education.*

I cannot do better than conclude this article by quoting (from a leader in the *British Medical Journal* of August 17th, 1889) the opinion of Professor Theodore Puschmann, of Vienna. He says, "The methods of the middle ages have held their own longest in the medical teaching of England. Therefore it happens now-a-days, even if seldomer than aforetimes, that medical students become apprentices to a practical physician. They remain with him an entire year, in order to obtain a superficial acquaintance with the things which life will one day require of them. In this method much depends upon the individuality of the pupil, and nearly everything upon the personality of the teacher. If the student is industrious and gifted, and if the teacher possesses knowledge and pleasure in the performance of his duties, then this year is to the former of inestimable advantage to his future studies. Under other circumstances, it is lost time, and serves at the most to provide him with a mechanical routine, which often borders closely on charlatanism." He speaks in similar terms of initial pupilage to a hospital. "There," he says, "the students believe that they will have the opportunity of observing many

* The comments on this case have for the most part been added. Only some of the remarks printed above were communicated to the students.

sick, and hope to receive from the house physician teaching on the more important phenomena. If they are not deceived in their expectation, they gain a certain dexterity and readiness in their intercourse with the sick, which is most useful to them in their later clinical and medical practice." In direct opposition, however, to Mr Wheelhouse's opinion, Professor Puschmann concludes, "that in many other directions this mode of entry into the medical profession is objectionable, It misleads the learner into superficiality, because it accustoms him to skim the reality of things, since knowledge and intelligence fail to enable him to reach the bottom. Moreover, the results obtained scarcely repay the sacrifice of time and trouble which they occasion the physician who acts as teacher, and still less justify the inconveniences in the treatment of disease which they bring in their train.'"

III.—Case of Herpes Zoster on the Hand.

My friend Dr Stewart, of Newport, sends me the following brief note of a case of herpes zoster on the hand:—

"I have recently seen a case of herpes zoster distributed over the pectoral muscle, down the inner side of the arm from the front of the axilla, on the forearm, wrist, palm of the hand, and on the palmar aspect of the first and second phalanges of the fingers. The pain in the palm of the hand was so intense, as to make me think that a deep abscess might be forming; but all subsided in the course of a few days. I have seen more cases of herpes zoster during the last three years, than in the previous dozen years."

IV.—The Value of Headache in the Diagnosis of Intracranial Tumours.

Of all the symptoms of intracranial tumour, headache is the most frequent; it is usually the first to attract attention, and is very frequently the symptom which is most distressing to the patient, and that which most urgently demands relief.

In the great majority of cases of intracranial tumour there is more or less headache. In some the pain is intense; the patient may describe it as "agonising," "unbearable," or may say he feels as if his "head would burst." The severity of the suffering may be so great as to suggest the idea of suicide.* As a result of this headache there is often sleeplessness and great exhaustion. In others, the headache is comparatively slight. In a small proportion of cases it is altogether absent. In young children—in cases in which the fontenelle is not closed—headache is, in my experience, frequently absent; but even in the adult, and in cases of large intracranial new growths, there may be no headache. In some cases, the pain is dull and boring; in others, shooting and neuralgic in character. In many, the headache is paroxysmal or subject to paroxysmal exacerbations; in others, it is more or less constantly present; in some, and more especially (but not exclusively), in syphilitic cases, the pain is worst at night.

In many cases, the paroxysms of headache are associated with vomiting or other signs of cerebral disturbance. In some cases, the patient is quite free from pain between the paroxysms. The length of the intervals is usually quite irregular; but in a few cases the paroxysmal exacerbations seem to be associated with menstruation, and to occur periodically every month.

Anything which deranges the cerebral circulation, such as hanging the head down, rising from the recumbent to the erect position, coughing, sneezing, laughing, straining at stool, &c is apt to aggravate the headache, or to induce a paroxysm. In many cases the headache is first when the patient first gets up in the morning.

The occurrence of headache and vomiting first thing in the morning is very suggestive of a cerebral lesion, but alcoholic dyspepsia and

* See a case of glio-sarcoma of the cerebellum, reported by Dr Leslie and myself, in the *Edinburgh Medical Journal* for January 1887, p. 591.

some other conditions, such as the vomiting of pregnancy and of uræmia, must be excluded.

The position of the pain is most variable It is usually described as "internal," but in some cases is external and superficial. In some cases it is referred to the whole head; in others, it is limited to one side, to the vertex, or to the frontal, parietal, or occipital region. In a certain proportion of cases, it is very limited, and localised; in many of these, tenderness on skull percussion, or even on gentle pressure over the affected spot, is present. In some, localised alterations in the bones of the scalp (such as thickenings or depressions, syphilitic nodes, &c.) can be felt at the point of tenderness. In some cases, in which the pain is neuralgic in character, it is limited to the area of distribution of a particular branch of the fifth nerve.

The causation of headache in cases of intracranial tumour is probably various.

The most common cause is probably increased intracranial tension, with resulting stretching of the membranes and irritation of (both as a result of stretching of, and pressure on) the sensory nerve fibres distributed to the membranes of the brain and the cranial parietes. If this is so, we should naturally expect headache to be most prominent and severe in those cases in which greatly increased intracranial pressure is quickly produced (*i..e*, in large, quickly growing tumours). When the increased intracranial pressure is slowly established, and more especially in those cases in which (in consequence of dropsy of the ventricles, a general œdematous condition or marked anæmia of the brain tissue) the cerebral sensibility is dulled, little or no headache may be complained of.

In some cases the headache is due to direct involvement of the membranes, periosteum or bones, by the tumour, or by the inflammatory changes produced in the immediate neighbourhood of the new growth, with consequent pressure on, or inflammatory irritation of, the branches of the fifth nerve which are distributed to the affected part.

The effect which rapid alterations in the cerebral circulation, such as are produced by sudden rising from the recumbent to the erect position, sneezing, coughing, &c., may have in aggravating the headache has already been alluded to. *Vice versâ*, the relief which is in some cases afforded by free purgation, the application of cold to the head, and the administration of remedies which reduce the arterial blood pressure, must be remembered.

In some cases headache is due to direct implication of the trunk of the fifth nerve, or of some of its larger branches; and in such cases the pain is referred (in accordance with the law of eccentric projection), not so much to the seat of the lesion, as to the area of distribution of the affected nerve.

In some cases, the headache of intracranial tumour is perhaps the result of a nerve storm, similar in character to that which produces the pain of ordinary sick headache or migraine. Such, at all events, was the late Dr Hilton Fagge's view. He writes—"Now my hypothesis is that a tumour or tubercle causes a transitory vertigo, or an epileptiform attack, or an attack of sick headache, in exactly the same way as any other disturbing agent. I conceive that the nerve storm so produced has exactly the same effect as when it is merely the result of over-fatigue, or irritation of the generative organs, or disorder of the stomach. And I think it is probable that frontal headache, even when unattended with the other characteristic symptoms of an attack of migraine, is yet very often of that nature. If this be granted, it ought to follow that pain in the forehead should point less directly to the anterior part of the brain as the seat of a tumour, than occipital pain to the cerebellum or posterior lobes. I do not know whether growths situated in any particular region are more apt than others to be accompanied by vertigo or epileptiform convulsions, as distinguished from those seizures which are limited to the muscles of certain parts, and to which I shall presently refer. But Dr Russell Reynolds was led, by the examination of a large number of cases, to the conclusion that convulsions in general were less common when the disease affected the ante-

rior lobes than when it occupied the posterior lobes or the cerebellum.*

The diagnostic value of headache as a symptom of intracranial tumour.—Headache is such a common symptom, and may be due to so many different conditions, both functional and organic, that *per se* it is not of much importance as an indication of serious organic cerebral disease (*e.g.*, tumour) unless it is very persistent and severe, and unless all the other conditions which may give rise to headache can be excluded.

But since it is the most frequent of all the symptoms of intracranial tumour, it is of great importance when associated with other general symptoms (vomiting, and more especially double optic neuritis) indicative of tumour, or when distinct indications of a localised cerebral lesion, such as localised spasms or paralysis, are present.

While it may be stated that in the absence of headache a *positive* diagnosis of intracranial tumour is seldom possible, it must be remembered that the absence of headache does not absolutely exclude tumour.

It must also be remembered that headache is often a prominent symptom in the very conditions which are most liable to be confounded with tumour—such as Bright's disease, plumbism, errors of refraction with anæmia, migraine, extracranial syphilis, meningitis, &c.

The localising value of headache is not as a rule great. Frontal headache more especially is apt to mislead the observer. In some cases, however, the pain corresponds more or less closely, and in a few cases most accurately, to the position of the tumour. In many subtentorial (cerebellar) tumours, the pain is chiefly referred to the back of the head; and in tumours of one hemisphere, it is not very uncommon to find the pain located on one (the same) side of the head. The localising value of headache is much increased if, in addition to the fact that the pain is confined to a limited area, there is also tenderness on percussion, or some other local alteration (such as a depression or swelling on the surface of the cranium) at the seat of the pain.

Localised pain, with tenderness on pressure, suggests very strongly that the tumour is superficial, and that the bone or membranes are affected. In syphilitic cases, in which the pain is often of this character, and indeed in all cases in which it is superficial and localised to one part of the cranial wall, it may of course be the result of an *extra*cranial as well as intracranial lesion.

In those cases in which the pain in the head is superficial and neuralgic in character, the fact that it is felt in the areas of distribution of *all* the branches of the fifth nerve makes it much more probable that it is due to central disease (*e.g.*, pressure on the nerve *within* the cavity of the cranium) than when one division only of the nerve is involved.

In illustration of the very definite manner in which pain and other abnormal sensations are sometimes limited to the exact seat of the intracranial tumour, the following statement of Hilton Fagge may be quoted:—"The museum of Guy's Hospital contains a large tumour, three inches in diameter, which I found many years ago in the left hemisphere of a girl, a patient of Dr Wilks, who suffered severely from pain in the head, and who had declared that when she turned her head to one side she felt something move in its interior. Another preparation consists of a small growth from the dura mater, taken by Dr Day, of St Neots, from an old woman who died of bronchitis. She had often expressed a wish that her head should be opened, because for years she had experienced anomalous pains in it, and a sensation of coldness at one spot, not larger than a shilling; this corresponded very nearly with the seat of the tumour, which was found after her death."*

* *Principles and Practice of Medicine*, vol. i., p. 526.

* *Principles and Practice of Medicine*, vol. i., p. 328.

Studies in Clinical Medicine.

FRIDAY, JANUARY 10, 1890.

CONTENTS.

I.—CASE OF SO-CALLED PERFORATING TUMOUR OF THE SKULL.

Boy; aged 4 years and 8 months.

(The patient had not been seen before he came into the Clinic.)

THE patient was profoundly cachectic looking; the left eyeball was markedly prominent, the conjunctiva and eyelids discoloured and ecchymosed; an elastic swelling, the size of a small orange, projected from the fore part of the vertex of the skull.

Dr B. (*to the Students*). This, gentlemen, is obviously a case of great interest and rarity. It is one of the few cases in which one can, at first sight, and without having asked the patient any questions, venture to give a positive diagnosis. I have little doubt that we shall find that it is a case of perforating sarcoma of the skull. Perforating sarcoma of the skull is one of the rarest forms of intracranial tumour. I have never had a case of the kind under my own care, but in my book on intracranial tumours, I have, through the kindness of my friend Dr Drummond, figured two remarkable examples of the disease, which were under his care in the Newcastle-on-Tyne Infirmary.

The mere fact that there is a tumour projecting from the surface of the cranium, would not, of course, warrant the diagnosis I have suggested. I base the provisional diagnosis, which I have made at first sight, on the combination of tumours which are evidently present in this case. The marked projection of the eyeball, when taken in combination with the presence of the elastic tumour on the top of the head is almost certainly indicative of a tumour in the orbit. Now, the presence of a tumour in the left orbit, and of another large tumour projecting from the skull, is to my mind conclusive evidence that we have to do with a case, in which there are multiple malignant growths. The strong probability is, that multiple malignant growths in these situations, and in a boy of this age, are sarcomata, and that the new growth is that form of sarcoma which produces perforation of the bones of the skull. (It would be more correct to say, which, when it affects the cranial bones, is apt to grow in both directions—inwards towards the brain and outwards towards the scalp—and which, in some cases, produces perforations in the bone. In this case, as the examination of the tumour after death showed, the bone was not perforated.)

Dr B. (*to the patient's mother*). How long is it since you noticed the lumps on the boy's head?

Patient's mother. Three weeks yesterday.

Dr B. How long has his eye been like that?

Patient's mother. Six or seven weeks. The eye became discoloured six or seven weeks ago. The left eye was always a little larger than the right, but during the last three weeks it has got much larger.

Dr B. You say that the left eye always looked larger than the right?

Patient's mother. Yes, it has looked larger than the right since his birth.

Dr B. (*to the Students*). The head is large.

Patient's mother. He always had a large head.

Dr B. Did you notice anything wrong with his head before the lumps appeared?

Patient's mother. No, there was nothing wrong with his head till three weeks ago.

Dr B. Was he quite well till three weeks ago?

Patient's mother. Yes. He was a strong healthy child. We noticed nothing the matter with him till three weeks ago.

Dr B. (*to the Students*). He now looks extremely ill; he is profoundly cachectic looking; the case is evidently running a very rapid course.

Dr B. (*to the Patient's mother*). What does he complain of?

Patient's mother. Great weakness. He is so weak he is unable to stand. He has no appetite; last night he had a severe bleeding at the nose; it has weakened him very much; he complains of headache.

Dr B. How long has he had the headache?

Patient's mother. Three or four weeks.*

FIG. 86.—Enlargement of the head in a case of so-called perforating tumour of the dura mater. (After Drummond.)

Dr B. Is the headache severe?

Patient's mother. Some days; he has had no sleep for a fortnight because of the pain in his head.*

Dr B. What was the first thing that he complained of?

Patient's mother. A sore head.

Dr B. What was the next thing?

Patient's mother. A cough, and his eye began to get black.

Dr B. Has he vomited?

Patient's mother. Yes.

Dr B. The illness did not begin with vomiting?

Patient's mother. No.

Dr B. Do you think he hears well and sees well?

Patient's mother. Yes.

Dr B. Does he seem to understand all you say to him? Is he as intelligent as he was before he became ill?

Patient's mother. Yes.

* The confusion in the mother's statement shows the great difficulty that there is in getting, unless with great care and repeated questioning, the exact facts of the case. She first stated that the discolouration of the eye was noticed six or seven weeks ago; she then stated that the headache preceded the discolouration of the eye; finally, she stated that the headache was first complained of three weeks ago.

On subsequent cross-examination it was ascertained, that the headache was of six or seven weeks' duration; that the lump on the head had been noticed three weeks before the patient came to the Clinic; and that, with the appearance of the tumour, the boy had become really ill.

* At the meeting of the Medico-Chirurgical Society, at which I showed the specimen, I erroneously stated that there had been no headache. During the last few weeks of

Dr B. Do you know if he ever got a blow on his head?

Patient's mother. I could not say.

Dr B. Are there any lumps on his belly, or on any other part of his body?

Patient's mother. No.

Dr B. Have you any other children?

Patient's mother. Yes.

Dr B. Are they all healthy?

Patient's mother. Yes.

On examination, no tumours were detected in any other part of the body. The cranial tumour was soft and elastic; it did not appear to be painful on pressure; the left pupil was somewhat larger than the right, and contracted much more sluggishly to light than the right; the right conjunctiva and right upper eyelid were discoloured, but the extravasation of blood was much less marked in the right than in the left conjunctiva.

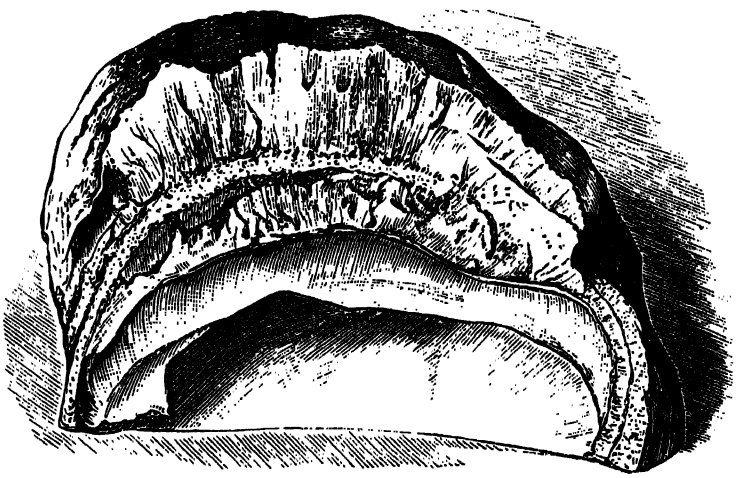


FIG. 87.—A section through the left and smaller half of the skull, and new growth in the case of perforating tumour of the skull, represented in fig. 86. (After Drummond.) The section was made just to the right of the falx; the right hand side of the figure corresponds to the frontal end.

Dr B. (to the Students). It will be advisable to examine the optic discs, and also to make a microscopical examination of the blood. In Dr Drummond's cases there was double optic neuritis.

The so-called perforating tumours of the dura mater have been sometimes described as perforating cancers. Mr Lawson Tait described some cases of the sort, and termed them, if I remember right, perforating cancers of the dura mater. Dr Drummond believes, probably with truth, that tumours of this kind are always, or, to speak cautiously, usually sarcomata. In his cases, the tumours were small round-celled sarcomata.

The profound cachexia in the case described in the text was a very striking feature, and suggested the advisability of examining the blood. The bone marrow is now known to be a very important blood-forming structure. Possibly in this case the profound cachexia may have been the result of a lesion of the bone marrow. Whether this was so or not I am unable to say, for the diseased frontal bone was the only one available for microscopic examination.

On subsequent examination, well marked optic neuritis was seen to be present in both eyes. Microscopical examination showed that the blood was very watery, that the white corpuscles were in considerable excess, while the red were diminished in numbers. Most of the red corpuscles appeared to be normal, as regards shape and size; the blood contained a large excess of debris and granular material, apparently of the same structure as the white corpuscles. A cultivation of the blood yielded negative results

I was kept informed as to the subsequent course of the case by my friend Dr Welsh of Kinghorn, who kindly sent the patient to the Clinic.

Dr Welsh writes me that the whole course of the case was twelve weeks. The first symptoms noticed were ecchymosis of the eyelids, pain in

the patient's life, the headache entirely disappeared. During the earlier stages of the case, as stated in the text, headache was a prominent symptom of the case.

the head, and restlessness. In the later stages of the case, there was absolutely no pain; the patient was quite conscious until five minutes before his death. Just before death he was able to recognise his mother's voice. Five weeks after the lump on the head was noticed, he became blind, and continued blind until the time of his death.

In Fig. 84, the prominence of the left eyeball and the tumour on the vertex are well shown. The drawing is an exact copy of a photograph which was taken on the day that the boy was brought to the Clinic. The drawing fails to give a true idea of the appearance of the patient. The marked cachectic appearance, which the patient presented, and the expression of profound debility and grave illness, which were such noticeable features in the case, are not in any way brought out, either in the photograph or the drawing which was made from it.

No case, which has come under my own notice, has impressed me more forcibly than this one has done with the importance of coloured drawings. Had this case been represented by a good coloured drawing, the impression produced on the mind of the observer would have been altogether different from that which is produced by the photograph or uncoloured drawing.

Fig. 84 shows a longitudinal vertical section through the tumour, frontal bone, and brain. The tumour, which is a small round-celled sarcoma, containing a large quantity of fibrous tissue, is situated on the exterior of the dura mater. It appears to have originated in the bone. A large mass of tumour tissue is situated, it will be seen, on the outer and inner aspect of the frontal bone. There was, however, no perforation of the bone, such as is usually present in cases of this description. Several smaller sarcomatous deposits were present, here and there, over the outer surface of the dura mater, covering the right hemisphere of the brain. I am unable to make any report as to the condition of the other cranial bones. The cavity of the orbit was not examined. Dr Welsh, who kindly made the post-mortem examination, and to whom I am indebted for the specimen, which is a very beautiful and typical one, unfortunately did not receive my letter, in which I requested him to examine the condition of the left orbit, until the morning after the autopsy.

I append some wood-cut illustrations, which Dr David Drummond has kindly placed at my disposal, and which show the appearance of the patients in his cases. (See Figs. 86, 87, 88.)

FIG. 88.—Perforating tumour of the dura mater. (After Drummond.)
The case is reported in the *British Medical Journal*, October 20, 1883, page 763.

II.—ABSCESS IN THE REGION OF THE KIDNEY.

Man; age 37; stonemason; June 5, 1889.

(The patient had not been seen before he came to the Clinic.)

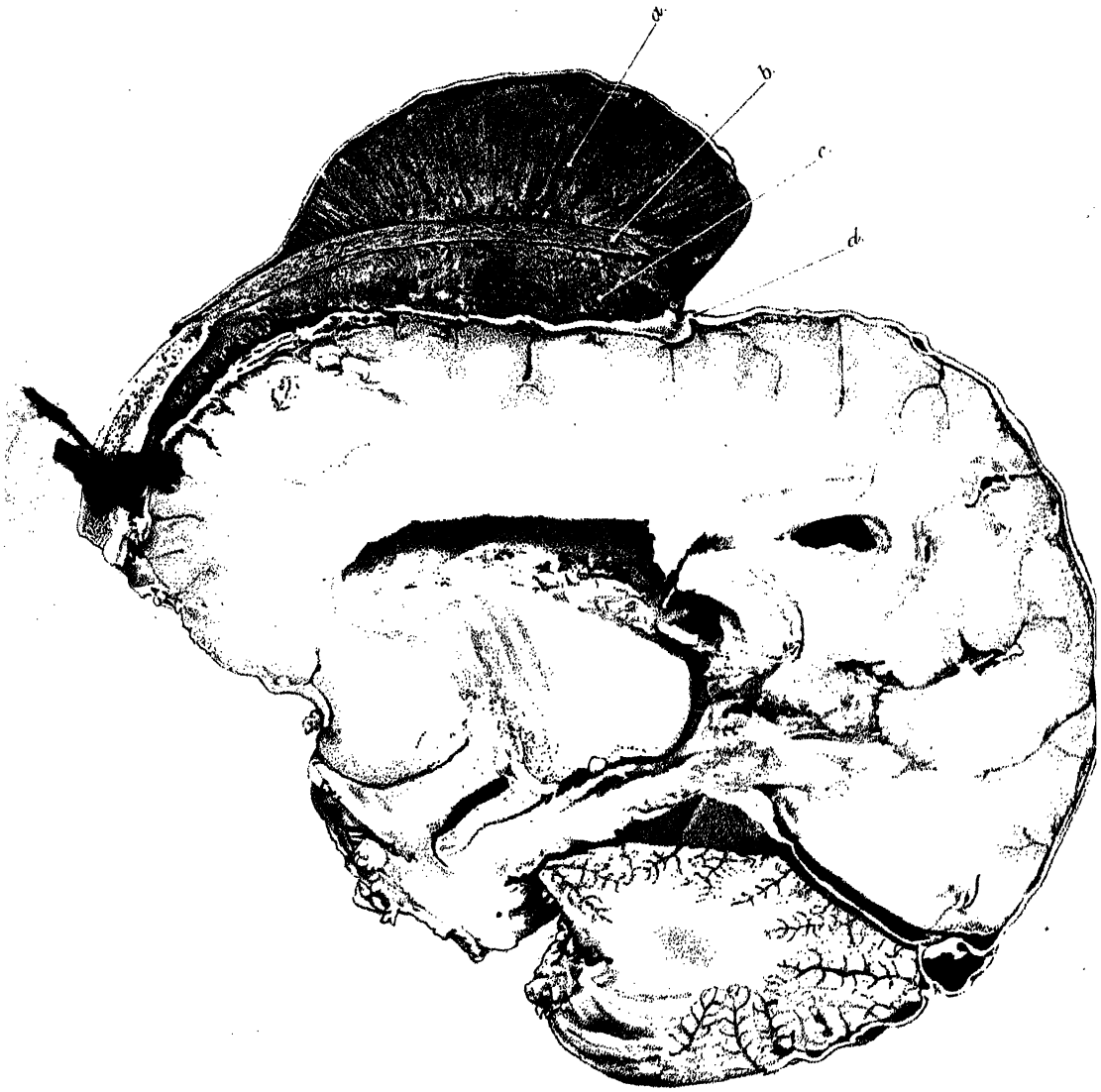
Dr B. What do you complain of?

Patient. A lump in my side, and my legs swell.

Dr B. How long have you been ill?

Patient. Three months.

FIG. 84.—Case of perforating sarcoma of the skull.
A tumour, the size of a large egg, is seen to be situated on the vertex. The left eye is prominent, evidently the result of a growth within the cavity of the orbit.



85.—Longitudinal vertical section through the frontal bone and brain in the case of perforating sarcoma of the skull represented in Fig. 84.

The new growth is situated on the exterior of the dura mater (*d*). The letter *a* points to a large mass of tumour substance which is situated on the outer surface of the frontal bone (*b*). The letter *c* points to that portion of the tumour which is situated between the under surface of the frontal bone and the dura mater (*d*).

The tumour, which is a small celled sarcoma, contains a large quantity of fibrous tissue. The new growth appears to spring from the bone or periosteum rather than from the dura mater. Though there is a large mass of tumour substance on each side of the frontal bone, the bone is not perforated.

Dr B. Do both of your legs swell?

Patient. Yes; they get quite black, they swell and get black when I walk a little distance.

Dr B. How far do you require to walk to make your legs swell and get black?

Patient. A hundred yards.

Dr B. Are your legs black at present?

Patient. Yes.

Dr B. Let us see one of the legs.

A little venous mottling was present on the legs, but the feet and legs were not swollen, and the legs were not black.

Dr. B. Do you complain of anything else?

Patient. No.

Dr B. Do you feel ill?

Patient. I have a little pain with it.

Dr B. Have you lost flesh? (The patient looked thin and ill).

Patient. I never eat anything.

Dr B. Have you the means of getting plenty to eat?

Patient. Yes.

Dr B. (the tongue was stained black). Have you been taking iron.

Patient. Yes.

Dr B. Go into the next room and take off your shirt, and let me see the lump in your side.

Dr B. (*to the Students*). The patient certainly looks ill, but he gives me the impression of exaggerating. There is certainly no swelling of the legs. We will see, when he comes back, whether there is any lump in his side.

One should be very careful in coming to the conclusion that a patient is shamming. It is, in many cases, a much more difficult thing to diagnose feigned disease, or to say that there is absolutely no disease, than to diagnose the presence of real or of actual disease.

I have heard of a student in a clinical examination being asked to examine a perfectly healthy person. Well, I don't myself think that is altogether fair to the student. When one finds a man in bed in a hospital, and especially when one is told, for the purposes of a clinical examination, to examine a patient who is in bed in a hospital, one expects that he is ill—that he has something very distinctly the matter with him. There would of course be no unfairness, if the candidates were told that some of the supposed patients had nothing the matter with them. That would be fair enough. But unless it is clearly understood, that some of the supposed patients are healthy, it is, I think, a little hard on the student to be sent to examine a supposed patient who is not a patient.

One of the great difficulties which both the students and the examiners have to contend with in every form of examination, but especially at a *vivâ voce* examination, is the nervousness of the candidates. It is, I consider, the duty of every examiner to place the candidates at their ease. No one who bullies and frightens the candidates is fitted to be an examiner. Kindness and courtesy are, I consider, absolutely essential requirements in a good examiner. The duty of an examiner is to find out what the candidate does know, and not to find out what he does not know. Students at examination times are very easily flurried. Many men who are really well up make a very poor appearance at a *vivâ voce* examination, simply because they become flurried, lose their heads, and, for the moment, forget facts with which they are in reality perfectly familiar. It is the duty of every examiner to endeavour to calm and allow for the nervousness of the candidates. A kind and courteous manner, the ability to put his questions in as plain and direct and understandable a manner as possible, together with a judicial mind, absolute impartiality, undeviating good temper, and knowledge up to date, are, in my opinion, the qualities which go to form a really good examiner. Teachers, as a rule, make the best examiners, for the simple reason, that they are *au courant* with the facts—with the knowledge—which the student is expected to get. It is not, however, every teacher who is fitted to be an examiner. It is in fact a difficult thing to be a really good examiner.

There is much more that I might say on the subject of examiners and examinations. Possibly I may return to the subject in some

future number of these *Studies*. What I wish now to say is this, that if a student is sent to examine a patient who is in bed in hospital, he naturally expects that the patient has something definitely and distinctly wrong with him which he ought to discover. If the supposed patient has nothing the matter with him, the student, failing to detect any evidence of disease, is very apt to become flurried, to lose his head, and to make a very much poorer appearance than he would have done if he had not become flurried. Any method of examination which is calculated, or likely, to flurry the candidate is, I think, to be deprecated. It is for this reason that I object to any candidate in a clinical examination being set down to examine a healthy person, unless it is clearly understood beforehand that he may possibly be asked to do so.*

After the patient had removed his shirt, he was brought back to the Clinic. A well marked fluctuating swelling was present in the region of the right kidney. The urine was clear, acid, of normal specific gravity; it contained neither blood nor albumen.

Dr B. (manipulating the swelling). Is it tender to the touch?

Patient. Yes.

Dr B. Have you had any shivering?

Patient. Yes—four or five attacks.

Dr B. What is this mark along the spine?

Patient. I was burned over the spine a year ago.

Dr B. Were you then complaining of pain in the spine?

Patient. Yes.

Dr B. Have you any pain when you straighten out your leg suddenly? (Dr Bramwell here forcibly flexed the thigh on the abdomen.)

Patient. Yes; some pain in the back.

The abdominal muscles were very resisting, and it was impossible to explore the spine, or to palpate the region of the right kidney, satisfactorily from the front.

Dr B. Have you felt any loss of power in your legs? Does either leg feel weaker than the other? Are the legs much weaker than the arms?

Patient. No. I feel weak all over.

Dr B. Have you felt any numbness in your legs?

Patient. No.

Dr B. (to the Students). There can be no doubt that this man is seriously ill. The impression which I formed, that he was exaggerating, was, you see, quite erroneous. One requires to be very careful in drawing conclusions of that kind. It is especially difficult to come to a conclusion that a patient is shamming or exaggerating, when you see him for the first time. Before committing oneself to such an opinion, it is very advisable, in many cases at all events, to make oneself acquainted with the mental temperament and character of the individual.

The patient is suffering from a fluctuating swelling in the region of the right kidney. What the exact connections of the fluctuating swelling are, I have not been able to ascertain. The general condition of the patient, and the fact that he has had repeated shiverings, are highly suggestive that the swelling is an abscess. The fact that he was burned over the spine a year ago, is suggestive that at that time he was suffering from caries of the spine. The presumption is, therefore, that the fluctuating swelling in the right lumbar region is an abscess, resulting from disease of the vertebræ. Before committing oneself positively to that diagnosis, it would be advisable to ascertain definitely, by means of a diagnostic puncture, that the fluctuating swelling contains pus, and also to make a thorough exploration of the abdomen, under chloroform. The resistance of the abdominal muscles to palpation is so great, that I find it impossible to make a satisfactory examination of the abdomen. This is just one of the cases in which the administration of an anæsthetic is likely to be useful. By relaxing the resistance of the abdominal muscles, by means of chloroform, one can make, in such a case as this, a much more satisfactory examination of the abdomen.

It would be well also to have the urine re-

* These remarks on examinations and examiners were not delivered at the Clinic.

peatedly examined. The specimen which has been examined, contains neither blood nor albumen; but in cases of pyelitis and renal tumour, the urine may at times be quite healthy. It is not sufficient, in such a case as this, to be satisfied with a single examination of the urine. In all cases in which a tumour is situated in the region of the kidney—or at all events, in all cases in which there is any reason to suspect that a tumour or abscess, which is situated in the region of the kidney, is connected with the kidney—it is advisable to examine the urine carefully and systematically from day to day. In cases of renal tumours or renal abscess, the urine which at one time was quite healthy, may at another contain blood or albumen.

I am disposed to think that, in this particular case, the fluctuating swelling is an abscess, and that it is connected with the spine rather than with the kidney.

The diagnostic puncture affords, in many cases, very valuable information. It shows us, in some cases, that our diagnosis is right; in others, that it is wrong. About a month ago, a sailor came to the Clinic, after most of you had left, complaining of a small painful swelling, situated over the lower ribs on the right side. He stated that the swelling, which was about the size of a pigeon's egg, had developed within a week. The swelling appeared to contain fluid. There seemed to me to be distinct fluctuation in it. I accordingly diagnosed it to be an abscess; and with the object of deciding the diagnosis, I punctured it with a hypodermic syringe. I failed, however, to get any pus. The needle of the syringe contained a little thick curdy material, which to the naked eye looked like thick cheesy pus. Dr Hugh Jamieson, who happened to be present, suggested that an incision should be made into the swelling, and, with my consent, he plunged a bistoury into it. He failed, however, to find pus. Whether the swelling was a gumma or a sarcoma, or a fatty tumour, I do not know. The patient refused to come into hospital, or to have anything more done in the way of treatment.

Now this case illustrates the mistakes which one sometimes makes. As I frequently tell you, all of us make mistakes. It is not a question as to who makes and who does not make mistakes. It is only a question as to who makes the fewest mistakes. Well, in this case, I made two mistakes. The first one was, that I mistook a solid tumour for an abscess. Well, that is not a very serious mistake. It was a mistake which any one might have made under the circumstances of this particular case. The patient stated, that the lump had developed within a week, there seemed to be distinct fluctuation in it, and some thick cheesy material, like curdy pus, was actually withdrawn by the hypodermic needle. It was natural to suppose, under such circumstances, that the swelling was an abscess. And granting that it was an abscess, the correct treatment was obviously to put a bistoury into it.

The second mistake which I made in this case was, I consider, a much more serious one, for it was a mistake in principle. I should not have given my consent to the supposed abscess being opened in this the medical side of the hospital, by a gentlemen who was not officially connected with the hospital. I should have sent the patient to the surgical department of the hospital. The opening of a small abscess is not a great surgical operation, but it is an operation. And in a great hospital such as this, it is, in my opinion, very desirable that the surgical work of the hospital should be done by the surgeons, and only by the surgeons. It is, I think, advisable that that principle should be strictly adhered to, not only in the hospital, but so far as possible in private practice. Well, in this particular case I frankly admit that in asking a gentleman, who had no official connection with the hospital, to open the supposed abscess, and in this, the medical side of the hospital, I made a mistake. One learns by one's mistakes, and I shall take good care that I do not make the same mistake again. In this particular case, the mistake was easily made, and was to some extent perhaps excusable; or, at all events, understandable. The hour was late, it was nearly two o'clock. I knew that the Surgeons had in all probability left the wards,

and that the House-Surgeon had probably gone to luncheon. The diagnosis seemed to me clear. The mere opening of an abscess is a very trivial matter; and although Dr Hugh Jamieson was not officially connected with the hospital, yet he had previously been so. I mention these matters, not with the object of showing that I did not make a mistake; but in order that you may see how easily mistakes of this kind may be made, and how it was that I came to make this particular mistake. The matter may seem to you a very trivial one. I have brought it before you, since it illustrates a principle. Physicians who meddle with surgical cases, and surgeons who meddle with medical cases are, I think, very likely to make mistakes. There are, of course, exceptions; but it is, I think, a good general rule, both in private and hospital practice, that the physicians should confine their attention to medical, and the surgeons to surgical cases.

III.—The Treatment of Typhoid Fever.*

Let us now turn to the treatment of the disease. And in considering the treatment of typhoid fever, I shall enter into considerable detail. In some respects typhoid fever may be taken as the type of a continued fever. Much that I shall have to say with regard to the treatment of this disease, is applicable to the treatment of other febrile diseases, more especially to the other continued fevers. If, therefore, I appear to occupy what may at first sight seem to you to be a disproportionate length of time in considering the treatment of typhoid, it will, in reality, be well spent time, for it will enable me to shorten the remarks which I shall have to make with regard to the treatment of some of the other febrile diseases which we shall afterwards have to consider.

The treatment of typhoid fever is a very important subject. The disease is common; it not unfrequently proves fatal; it cuts short some of our most useful and valuable lives; and, as I have just pointed out, the treatment and management of a case of typhoid are in many respects illustrative of the treatment and management of the other continued fevers.

Now, in considering the treatment of typhoid, we must remember, in the first place, that the disease seems undoubtedly to be due to the introduction into the body of a definite, specific, organic, and particulate poison.

The first indication for the rational treatment of typhoid fever is, therefore, to neutralise this poison; to destroy it after it has gained access to the body; to prevent it producing its injurious effects on the system; or what in the end amounts to the same thing, to strengthen the resisting power of the tissues, so that they will, as it were, satisfactorily resist and overcome the invading host of typhoid germs.

Unfortunately, up to the present time we do not know of any satisfactory, safe, and certain means of destroying the typhoid germ during the period of its incubation in the system, and of so preventing the manifestation of the disease once the poison has been introduced into the human body.

Dr Theodore Cash, of Aberdeen, has made some very important experiments and observations on the prevention of some of the diseases due to germ poisons. He seems to have definitely and certainly determined that the administration of corrosive sublimate does in the lower animals prevent or modify the action of some of the germ poisons. He thinks it probable that in some of the acute febrile diseases, such as typhus fever, which are in all probability due to the introduction into the body of specific germ poisons (though in all cases the exact nature of the poison has not as yet been satisfactorily demonstrated), the administration of corrosive sublimate, during the period of incubation, may perhaps altogether prevent, or, at least, modify the severity of the subsequent attack.

* This article consists for the most part of a verbatim shorthand report of a portion of my systematic course of lectures on the Principles and Practice of Medicine. It is my intention to publish from time to time in these *Studies* portions of my systematic lectures, more especially those portions which relate to treatment.

When I come to speak of scarlet fever, and some of the other exanthemata, I shall have to tell you that some authorities believe that boracic acid, sulpho-carbolate of sodium, carbolic acid, and some other remedies of a similar kind, are undoubtedly useful for the same purpose. The experiments of Dr Cash seem to show, that corrosive sublimate is one of the most active and important of these remedies. So far as I know, corrosive sublimate has not been employed with the object of preventing the development of typhoid fever. The disease differs from typhus in as much as it is not directly contagious; the typhoid poison may, as we have already seen, be introduced into the system in a variety of ways (water, milk, air, &c.), but the fact that the poison has been introduced, and the date at which it was introduced, are almost invariably unknown to the patient. In the great majority of cases of typhoid, the patient is entirely unaware that he has even been in the way of getting typhoid until the disease actively declares itself with the usual invasion symptoms. Except in very rare instances, therefore, there is no opportunity of administering a remedy to prevent typhoid, even if we were in possession of such a remedy. As a matter of fact, we know of no satisfactory remedy for the prevention of typhoid. I would have no hesitation in giving corrosive sublimate to a person who had been exposed to typhus, with the object of preventing the development of the disease; but I should certainly hesitate to administer corrosive sublimate with the object of preventing typhoid. In order that this drug may exert its destructive effects upon the germ poison, it has to be given in considerable quantity. Professor Cash informs me, that if possible two grains of corrosive sublimate should be introduced, not of course all at once, for that would poison the patient, but in divided or frequently repeated doses, into the system of a patient who had been exposed to typhus. A sixteenth, twelfth, or eighth of a grain might, according to the circumstances of each individual patient (age, sex, &c.), be given every few hours, with the object of rapidly saturating the system with the drug. But it must be remembered that in rapidly saturating the system with corrosive sublimate, gastro-intestinal irritation and diarrhœa are very apt to be produced. Now, in the treatment of typhoid, it is very important, as we shall presently see, to avoid anything which is apt to irritate or produce inflammation of the small intestine. It is for this reason, that I should hesitate to administer corrosive sublimate with the object of preventing the development of an attack of typhoid. In most cases of typhoid, the question of arresting the development of the disease by the administration of drug remedies during the period of incubation, is, for the reasons I have already given, a theoretical rather than a practical one. I need not, therefore, occupy any further time in its consideration. I pass on to the much more important and practical subject, the treatment of a case of typhoid, after the disease has actually developed.

When the disease has actually developed, we do not know of any remedy which can be safely given, and which will, without risk to the patient, certainly and satisfactorily destroy the germ poison, which is the cause of the disease. The poison is probably contained in great abundance in the local lesion in the inflamed and ulcerated intestine.

If this point be admitted, it is of the greatest importance to realise, in the next place, that typhoid fever is a disease which tends, in the great majority of instances, to run through a definite and distinct course; and since we cannot cut the disease short by any therapeutic measures or drug remedies with which we are at present acquainted, our efforts must be directed to enabling the patient to tide over the period during which the attack usually lasts. If we can maintain his strength and enable him to tide over the period during which the disease usually lasts, and if there are no unusual accidents or complications, the strong probability is that he will recover. It is necessary to remember, in connection with this indication for treatment, that, as regards its duration, typhoid fever is much more variable than some of the other acute febrile diseases. In typhus, for example,

once defervescence occurs, convalescence is quickly established, and health rapidly regained. The reverse is often the case, as everybody knows in typhoid. This is one of the reasons which makes it very difficult to estimate the exact duration of some cases of typhoid. Another reason is, that individual cases differ very materially in their severity, and also in their exact duration. As I have already pointed out, the ordinary book descriptions—the usual type-pictures—of typhoid fever, are very often departed from in actual practice. For these reasons, it is consequently in many cases difficult to predict exactly the length of time the disease will last; to say how long the patient will have to be kept going, in order that he may be enabled to tide over the usual period of the disease, and to pull through the attack.

In treating cases of typhoid fever, we have to be prepared to meet the dangers which are apt to arise, and to do all we can to prevent those complications and accidents, which we know, as the result of experience, are apt to occur in the course of the disease.

Some of the chief causes of danger in typhoid fever are, failure of the heart, excessive diarrhœa, excessively high temperature, the retention in the blood of excrementitious matters and retained urinary products; while two of the most important accidents or complications are, perforation of the intestine and intestinal hæmorrhage.

It is of the utmost importance to recognise these risks and dangers, to look out for them, to do what we can to prevent them, and treat them as soon as they do occur.

There can, in my opinion, be no question that judicious management and treatment, do most materially modify the result in many cases of typhoid. Proper management and satisfactory treatment do, I believe, exert a very distinct influence upon the mortality of this disease.

There are, of course, in typhoid, as in all of the contagious and infectious diseases, some cases so mild that no treatment is required. There are other cases so severe that all treatment is useless. But between these two extremes, there lies a great mass of cases in which the disease is more or less, and in some cases very, severe, and in which without doubt judicious treatment, and careful feeding and nursing go far to decide, in many instances at all events, what the result shall be—whether the patient shall or shall not recover.

In connection with the treatment of typhoid, other points which should be kept in remembrance are, that the characteristic local lesion consists of an inflamed and ulcerated condition of the small intestine; and that, from the active onset of the disease to the termination of the attack—practically, we may say, till the cicatrisation and healing of the intestinal ulceration is completed—there is more or less febrile disturbance. During the active period of the disease—the first three weeks of the attack—the fever is high, and, as I have already pointed out, in typical cases it presents a very definite mode of development and course. (It is important to remember, that in many cases of typhoid, this course of the fever curve is by no means typical and characteristic; here, as in so many other diseases, the clean-cut and sharply drawn features of typical cases, as described in the lecture room and in the text-books, are very frequently departed from.)

The inflamed and ulcerated condition of the intestine, and the resulting tendency to diarrhœa, constitute one of the difficulties in the treatment of typhoid. In treating cases of typhoid, it is very important to remember this tendency to diarrhœa, and to avoid administering anything, either in the form of food or medicine, which will irritate the inflamed and ulcerated intestine. It is well known that the administration of even a mild purgative at the commencement of a case of typhoid, is apt to be followed by severe diarrhœa; and that in those cases of typhoid, in which powerful purgatives have been given at the beginning of the attack, the subsequent course of the disease is often very severe, and the diarrhœa very marked and intractable.

In typhoid fever, where the febrile process and the disease are of such prolonged duration, the administration of abundant nourishment is one of the most important indications for treatment;

but in attempting to carry out this indication, it is, as I have just mentioned, all important to remember the inflamed and ulcerated condition of the intestine; and to take care that the food, which is administered, is not calculated to increase the diarrhœa and to irritate the inflamed and ulcerated gut.

These are some of the general principles and fundamental facts which it is essential to bear in mind in considering the management and treatment of a case of typhoid.

The main object of a scientific and rational plan of treatment of an actual case of typhoid would be, as I have already mentioned, to neutralise or destroy the poison, and so cut short the disease. We have seen that, in the present position of medical science, we know of no remedy which is capable of fulfilling this indication—of destroying the typhoid germs, once they have actively developed in the system;—in other words, we know of no method of treatment by which we are able with safety and with certainty to cut short the attack, once the disease has actively developed and declared itself.

In treating cases of typhoid, we have therefore to fall back upon our second line of defence—clearly recognising our inability to cut short the disease, we have to concentrate our efforts in the endeavour to enable the patient to tide over the attack. Our object is to do all we can to enable him to satisfactorily battle with the disease.

We endeavour to carry out this indication in various ways and by various means. Our object is to sustain the patient's strength by every means in our power, to husband his resources and reserve as carefully as possible.

In order to carry out this indication, we have to see that he is, if possible, from the very commencement of the attack, placed in the best possible condition as regards his surroundings; that he is judiciously and carefully fed; that he is carefully nursed; and that the medicines and remedies, which are required in his special case, are regularly administered.

But it will be necessary to consider these and the many other points connected with the treatment of typhoid (such, for instance, as the administration of stimulants, the administration of antipyretics, the treatment which is required for the various complications, such as hæmorrhage, and perforation) individually and in detail.

In every case of typhoid, however mild the attack may seem to be, the patient should, from the moment when the disease is first recognised, be placed in bed. He should be kept in bed throughout the whole period of the attack, and on no account allowed to get up, not even to evacuate the contents of the bladder or rectum.

It is essential to see from the first that he is so far as possible kept at absolute rest in bed; that he has nothing to disturb either his body or his mind; to see that all sources of intestinal irritation are so far as possible prevented—nothing should be administered to him which is likely to irritate the intestine, to aggravate the intestinal inflammation, or to excite diarrhœa. During the earlier days of the attack, when he is conscious and acutely suffering, no efforts should be spared to relieve his sufferings, and to keep him in as cheerful and hopeful a frame of mind as the circumstances of the case will allow. There can, I think, be little doubt that, in some cases, the mental condition of the patient does undoubtedly influence the result, and in some measure at all events does determine whether he shall or shall not recover.

We all know what an important influence the nervous system exerts upon the processes of nutrition, growth, and repair; how the mind, to speak in popular language, acts upon the body, and the body on the mind; how, when the mind is depressed and filled with gloomy forebodings and fears, the functions of all the great viscera are apt to be disturbed or performed in a languid and sluggish manner. How, the whole vital tone, so to speak, is apt to be deteriorated, the resisting power of the organism weakened, contagious and infectious diseases (cholera, for instance) more likely to be contracted, and the processes of repair and recovery from disease, which is already present, seriously interfered with, or altogether pre-

vented; how easily persons of a cheerful, hopeful, and sanguine disposition of mind throw off their ailments; how, on the one hand—and this is the point to which I wish more especially to refer in connection with our present subject, the treatment of typhoid—a patient who firmly believes that he will get well, often does get well, even in apparently hopeless circumstances; and how, on the other, a patient who is impressed with the idea that he will die, often does die, even when we might reasonably have expected recovery to have taken place.

All experienced observers are at one in thinking, that in those cases in which the patient goes about during the earlier period of an attack of typhoid, the subsequent course of the disease is apt to be severe. There can also be little doubt that any violent exertion and sudden movement is apt, in the later stages of the disease—once the process of ulceration is fully developed—to lead to the production of perforation and perhaps in some cases also of intestinal hæmorrhage.

In the earlier stages of the attack, there is not, of course, the same risk of rupture of the gut or of intestinal hæmorrhage being produced by movement and exertion; but it is well to remember, that in some cases both the history of the exact duration of the attack, and the appearances indicative of the stage of the disease, are deceptive. It is not always possible to say with absolute certainty, even when the symptoms are apparently very slight, that intestinal ulceration has not already developed.

For these reasons, therefore, it is very desirable that persons who are suffering from typhoid fever should from the first be confined to bed, and that the importance of rest, and the necessity of avoiding any sudden effort (such as straining at stool, getting up to make water, &c.), should be insisted upon.

It is important, therefore, as soon as this disease is suspected, and still more, once it has been clearly recognised, that the patient should not only be sent to bed, but that he should be kept in bed.

When the patient is an adult, he should, from the first, be plaeed on a water bed or water mattress. In dealing with typhoid, we are dealing with a disease which lasts for several weeks, and in the later stages of which there is a marked tendency to the formation of bed sores. It is essential, if possible, to prevent the formation of a bed sore, for in some cases, at all events, the presence or absence of a bed sore is one of the conditions which determines the result. A bed sore is always a serious complication, and no effort should be spared to prevent its formation.

In the case of young children, a water bed is not so necessary, for they can be moved about so much more readily; and as a matter of fact, during a severe illness, such as an attack of typhoid, a young child passes a great deal of its time in the arms of its mother or its nurse. But in the case of older persons, more especially heavy adults, it is extremely desirable to place the patient on a water bed from the very first.

The bed itself (the bed on which the water mattress is placed) should not be too broad. A very broad bed presents great difficulties to satisfactory nursing. The bed should not of course be so narrow that there is a risk of the patient tumbling out of it; but it should be sufficiently narrow to allow of his being easily and comfortably reached by the nurse from either side, when he lies in the middle of it. A very high bed also interferes with the satisfactory nursing of the patient, just as a very broad one does. The bed should, therefore, be of a satisfactory height and width. These may seem small points, but it is just these small points which are often of the greatest practical importance; it is also these apparently small, but, in reality, very essential practical points, which are almost invariably omitted from the text-books. I shall, therefore, make no apology, either now or in the future, for referring to any point, however commonplace it may at first sight appear, provided only it has a direct practical bearing on treatment. Small points of merely theoretical importance are much better omitted from a course of lectures such as this. In practice, we want just as much theory as is necessary; but we want all the practical knowledge we can get.

Not that I, by any means, despise theory. Quite the contrary. Theories are often most useful and helpful. But the student's life is short; the amount of knowledge which he has to acquire is very great. The practical knowledge is absolutely essential. *It* should at all events be acquired. I am anxious in this course of lectures to give you as much practical knowledge as possible. The sort of knowledge which I wish you, in the first place, to gain from this course of lectures, is the knowledge which you will find of everyday use to you in your future career as practitioners of medicine.

The room in which a patient who is suffering from typhoid (and indeed every other acute febrile disease), is placed, should, if possible, be large and airy; in any case, whatever its size, care should be taken that it is well ventilated. For the maintenance of satisfactory ventilation, there is nothing like a fire on the one hand, and an open window on the other. Care must of course be taken that the fire is not too large, that the room does not get too hot, and that the patient is not exposed to any draught. If the room is small, and the weather warm, even a very small fire may be too much. But in summer time, and in warm weather, there is no difficulty in ventilating a sick-room by means of open windows. There is a very general impression amongst the laity, and it is, I fear, far too frequently shared in by the profession, that to open the window of the sick-room in which a patient who is suffering from a febrile or inflammatory disease is placed, is apt to be attended with danger. It is thought that the patient will catch cold. Such fears are groundless. The idea is a complete mistake. Provided that the patient is not exposed to a draught, he will not suffer from the fresh air. Indeed, quite the contrary, he will benefit by it. There need therefore be no hesitation, whenever the weather is suitable, in opening the window of the sick-room, provided always that a draught of damp or cold air is not allowed to play upon the patient. In summer time, and in warm weather, it is, in many cases, advisable to keep the window continually open from the top. Free ventilation of the sick-room is eminently desirable. In my opinion, an over-heated and badly ventilated sick-room is likely to be much more injurious to a patient suffering from typhoid fever, than an open window.

The room should be kept cool; it is desirable that the temperature should, so far as possible, be maintained at the same point; it is better to have it rather too cold than too hot. The temperature ought not to exceed 60° Fah. Both during the day and during the night it should be kept at or about 60° Fah. There should be no hesitation in opening the windows in summer, and in allowing, both in summer and winter, a plentiful supply of fresh air to enter the sickroom. The essential requirements are, that the room should be well ventilated, that its temperature should, so far as possible, be steadily maintained at the same height (at or a little below 60° Fah.), and that the patient should not be exposed to draughts. Provided these essential points are attended to, the more fresh air the patient gets, the better.

It is desirable to see that the patient is not covered up with heavy bed-clothes. Provided that he is kept covered, the lighter the bed-clothes are, the better. Patients who are suffering from high fever, instinctively throw off the bed-clothes, and allow the excessive heat of their bodies to be dissipated into the surrounding atmosphere. This is one of nature's indications for treatment, which it is well to bear in mind. I am satisfied that, not only are fever patients made uncomfortable, but that grave injury is often done by covering them up with blankets and heavy bed-clothes. In a case of typhoid fever, a single blanket, and in many cases a single sheet, is all that is required in the way of bed-clothes.

The point then which I have just been endeavouring to impress upon you is this, that in the treatment of a case of typhoid, and indeed of any other acute febrile disease, it is very important to see that the surroundings of the patient are, so far as possible, satisfactory. It is essential that from the first he should be made as cheerful and comfortable as possible, and that he should be placed in the best possible position, as regards his surroundings.

The next point—and it is one of the most important points in the treatment of a case of typhoid fever—is to see that the nursing arrangements are satisfactory.

In typhoid fever, we have to deal with an acute disease, which, in cases of ordinary average severity, is of several weeks' duration. In severe cases which recover, the most assiduous nursing is required for four, five, or six weeks; and in many cases, for a much longer period of time. In a severe case of typhoid fever, perhaps more than in any other diseased condition (I refer to medical, not to surgical or gynæcological cases), good nursing is essential. In many cases, the success of the treatment is more largely due to the nursing which the patient receives than to anything else. In many bad cases of typhoid, the kind of nursing which the patient receives, determines the result. Good nursing will often enable a patient to pull through a bad attack of typhoid. I feel perfectly convinced that, in many cases of severe typhoid, in which the patient who has been well and assiduously nursed just pulls through—to use a popular but very forcible expression, just pulls through by the skin of his teeth—that he would have succumbed to the disease, had he been badly or carelessly nursed. If I were myself to be so unfortunate as to be attacked with typhoid, I should like, above all things, to feel assured that I would be well and carefully nursed. In my opinion, good nursing is, in a case of typhoid, quite as important, I am not sure but I should not say even more important, than good doctoring. I do not in any way wish to minimise the importance of good doctoring in this disease. Far from it. Skilful and judicious doctoring can do a great deal for a case of typhoid, while—and this is perhaps still more important to remember—bad and injudicious doctoring can do a great deal of harm. If I were myself attacked with typhoid, I should take very good care that I had a good and judicious doctor. But what I do want to emphasise in the strongest possible way is, the importance of good and judicious nursing.

In arranging, then, for the treatment of a case of typhoid fever, one of the first points which the medical man has to attend to, is to see that there is a sufficient supply of good and capable nursing.

The patient must be well and carefully nursed during the whole period of the disease, during the night as well as during the day. In severe cases, the disease lasts, as we have seen, for several weeks, and careful nursing is in many cases required during the earlier weeks of convalescence. One nurse is quite incapable of nursing an adult affected with severe typhoid, satisfactorily. Two nurses are absolutely necessary—one for the night, another for the day.

Whenever the circumstances of the patient admit of it, two properly trained professional nurses, who have been accustomed to nurse typhoid cases, should be procured.

In this, as in every other disease, the female relatives or friends of the patient are often anxious to undertake the nursing. Now, speaking generally—and granting, of course, that thoroughly satisfactory and competent professional nurses can be procured—properly trained and skilled professional nurses, who have been accustomed to deal with the disease, are, in the case of typhoid, to be preferred to amateurs.

The loving zeal and affectionate solicitude which a mother or a sister or a daughter feels for the patient, are qualities, the importance of which, from a nursing point of view, it is impossible to over-estimate; but no amount of love, zeal, or affection, can supply the skill, experience, and knowledge, which are desirable in a nurse who has to deal with a bad case of typhoid fever.

It is a mistake to suppose that all women are "born" nurses. Some doubtless are. Most women doubtless possess the fundamental requirements which are essential for the making of a good nurse. Most amateurs would doubtless, after a little training and experience, be quite as able to nurse a case of typhoid, as the majority of professional nurses. Many amateurs would doubtless, after a little training and experience, be better than the average run of professional nurses. In some diseases, the amateur nursing of a loving wife or sister, is all that is required;

and when it is all that is required, it is, in my opinion, the best nursing that can be got. But in typhoid fever, a professional nurse is, I think (I speak generally— I do not mean to say that there are no exceptions)—provided that she is not only a well-trained and skilful nurse, but a right-minded, well-meaning, and zealous woman —to be preferred to an amateur.

And here it may be well, perhaps, to pause for a moment, to consider what are the qualifications of a good nurse and what are the duties which a nurse is called upon to perform in a case of typhoid fever.

(The subject will be continued in the next number of the *Studies*.)

IV.—The Symptoms and Signs indicative of an Intracranial Tumour.

If I may judge from my own experience, intracranial tumours are by no means so common in Edinburgh as in Newcastle. I am in the habit of accounting for this difference by the fact, that syphilis and head injuries are more common in Newcastle than they are here. Injury to the head is undoubtedly, I think, an important factor in the production of some forms of intracranial tumour. I believe that gliomatous tumours not unfrequently have their starting point in an injury to the skull. I feel satisfied, too, that scrofulous tumours in the cerebellum, in some instances, appear to result from, or, to speak more accurately, to follow a blow on the head. Perhaps a head injury may also, in some cases, act as an exciting cause in the production of sarcomatous and cancerous tumours.

Now what are the symptoms and signs indicative of an intracranial tumour? We are in the habit of dividing the symptoms and signs indicative of an intracranial tumour into two classes and groups. The "*general*" symptoms and signs, on the one hand, and the "*localising*" or "*focal*" symptoms and signs, on the other. The division is an important one, since it has a direct bearing upon diagnosis and treatment. It is, too, a real division, since it has not merely originated in the imagination of the lecturer or writer, but is founded on clinical fact. In many cases of intracranial tumour, there are no localising symptoms; the general symptoms and physical signs are the only symptoms and signs which are present.

The more important "general" symptoms and signs indicative of an intracranial tumour are, headache, vomiting, and double optic neuritis. In the great majority of cases of intracranial tumour, these symptoms and signs are present in combination. In many of the cases in which these three main symptoms—the chief, the most characteristic symptoms—of intracranial tumour are present, there are also, of course, other symptoms, such as spasms or paralysis, which have a definite localising value.

Headache alone, vomiting alone, or even double optic neuritis alone, is not indicative of an intracranial tumour. But when headache, vomiting, and double optic neuritis are met with in combination, the presence of an intracranial tumour should always be strongly suspected, for an intracranial tumour is the condition which is most frequently the cause of these symptoms.

Headache, vomiting, and double optic neuritis, in combination, may be, of course, due to other conditions than a tumour within the cranium. Meningitis is one of these conditions, kidney disease is another, and lead poisoning is a third. As a rule, it is easy to exclude meningitis, kidney disease, and lead poisoning. In most cases in which headache, vomiting, and double optic neuritis are due to meningitis, there is fever (pyrexia). It is rarely that meningitis without fever (chronic meningitis) gives rise to headache, vomiting, and double optic neuritis, except when the meningitis is syphilitic (and then there is usually a gumma), or except in those cases in which the meningitis has its starting point, its exciting cause, in a localised "coarse" or naked eye lesion, such as a tumour.

When a tumour and meningitis are both present, it may, of course, be very difficult, in

fact in some cases it may be impossible to say, whether the symptoms—the headache, the vomiting, and the double optic neuritis—are the result of the tumour or of the meningitis, which is (usually) secondary to the tumour. The matter is not perhaps of very great practical importance, for the treatment of the two conditions is very much the same. If iodide of potassium in large doses, or failing the iodide alone, mercury along with iodide of potassium, does not effect a cure, no drug remedies are likely to give relief. The important practical fact to remember is that when headache, vomiting, and double optic neuritis are associated with other symptoms (negative or positive) suggestive of chronic meningitis, that the meningitis is usually either syphilitic or secondary to a "coarse" cerebral lesion—such as a tumour or abscess.

I shall not on the present occasion enter into the differential diagnosis of cerebral tumour and cerebral abscess. That is a very interesting and important practical subject, to which I hope to refer in some future number of these *Studies*. All I need say at present is this, that the differential diagnosis of cerebral tumour and abscess is usually impossible unless some one or other of the usual causes of cerebral abscess—such as ear disease, nose disease (in short, disease of some one or other of the cranial bones), a localised injury to the skull, or lung disease, likely to be followed by secondary abscess in the brain—is present. Fortunately for the purposes of correct diagnosis, a cerebral abscess very rarely indeed occurs without some definite and distinct exciting cause (such as ear disease, head injury, etc.). If, therefore, there is no obvious cause of cerebral abscess, the presence of headache, vomiting, and double optic neuritis, in combination, would be indicative of some other form of "coarse" intracranial disease (than abscess)—in other words,—granting that meningitis, kidney disease, and lead poisoning could be excluded—would be indicative of an intracranial tumour.

As a rule, it is, of course, quite easy to exclude kidney disease and lead poisoning.

The examination of the urine and of the heart and vascular system enables us to determine the presence or absence of kidney disease. I have referred to this subject in a previous number of these *Studies*. (See page 38.) I need not again enter into details.

In the cases of lead poisoning in which headache, vomiting, and double optic neuritis are present, there is usually (invariably in my experience) a blue line on the gums, and generally a history of constipation, colic, and other symptoms indicative of plumbism. The occupation of the patient is also a point of great diagnostic importance. I do not, however, on the present occasion propose to enter into details, with regard to the interesting subject of lead poisoning. I shall return to it again in a future number of these *Studies*. The nervous symptoms which may be produced by lead poisoning are very numerous, and the space which I have at my disposal in the present issue is not sufficient to allow me to consider the subject as I would like to consider it.

Returning to the subject of intracranial tumours, I repeat that when headache, vomiting, and double optic neuritis are present in combination, a positive diagnosis of intracranial tumour may be given, provided that meningitis, cerebral abscess, kidney disease, and lead poisoning can be excluded.

I should add that in some cases of profound anæmia, the symptoms and signs of an intracranial tumour are more or less closely simulated.

I should also say that in some exceptional cases, headache, vomiting, and double optic neuritis, are the result of a diffuse cerebritis, and not of tumour. This peculiar form of cerebritis, which it is impossible to distinguish clinically from an intracranial tumour—for the symptoms and signs may be identically the same in the two conditions—is, however, exceedingly rare. So rare that, for the purposes of diagnosis, it may practically be left out of account.

The *localising* or "*focal*" symptoms will be referred to in the next number of the *Studies*.

Studies in Clinical Medicine.

FRIDAY, JANUARY 24, 1890.

CONTENTS.

I.—THE SYMPTONS INDICATIVE OF AN INTRACRANIAL TUMOUR.

(Continued from page 264.)

IN the last number of these *Studies*, I stated that the symptoms which result from the presence of an intracranial tumour may be divided into two main groups—the "*general*" symptoms on the one hand, and the "*localising*" or "*focal*" symptoms on the other.

The more important of the "general" symptoms are, as we have seen, headache, vomiting, and double optic neuritis.

When these three great symptoms are met with in combination, the presence of an intracranial tumour should always be suspected. Provided that meningitis, kidney disease, lead poisoning, cerebral abscess, and the extremely rare condition, diffuse cerebral sclerosis, can be excluded, the presence of headache, vomiting, and double optic neuritis in combination, may be held to be indicative of the presence of a tumour in some part of the cranial cavity. It is perhaps going too far to say, that no other diseased conditions, except those which have just been enumerated, are capable of producing headache, vomiting, and double optic neuritis. Profound anæmia is undoubtedly in some cases attended with double optic neuritis, and with some degree of headache; and possibly, in some cases of profound anæmia, vomiting may be present as an accidental or associated symptom. But the presence of very profound anæmia would put the physician on his guard, and would necessarily make him hesitate in committing himself to a positive opinion. In some cases of leucocythæmia, too, in which the nerve centres are the seat of profound vascular alterations and of numerous hæmorrhages, headache, vomiting and double optic neuritis are present. In cases of this description, the physician has to ask himself whether the vascular alterations which are presumably present in the brain, are sufficient to account for, or likely to account for the symptoms—the headache, the vomiting, and the double optic neuritis. The same question has constantly to be answered in dealing with diseased conditions. It is easy to lay down general rules for diagnosis or differential diagnosis in a text book or in a lecture room; but every one who has had much experience in dealing with disease as it occurs in the living patient, knows how frequently the text book descriptions of disease and the text book rules for diagnosis and differential diagnosis are departed from and have to be modified at the bedside. There are indeed few symptoms, or even combinations of symptoms, which are pathognomonic and actually indicative of any single diseased condition. In dealing with disease as it occurs in the living patient, we are constantly meeting with exceptions and anomalous conditions. Any well informed student is able to recognise and diag-

nose the typical cases, which conform to the ordinary text book descriptions; but it is only the man of wide knowledge and extensive bedside experience who is able to recognise the exceptional and anomalous cases.

The various conditions which we group together under the common term intracranial tumours, form no exception to this general rule. In some cases of intracranial tumour, there is no headache; in others, there is no vomiting; in others, again, there is no double optic neuritis. It is seldom indeed, though it does sometimes actually occur, that all of these symptoms are conspicuous by their absence; while in most cases of intracranial tumour, all three of them are present in combination, or at all events, are observed at some period or other of the case.

Double optic neuritis is by far the most important of all the symptoms of intracranial tumour.

Firstly, Because it is an objective sign, which does not depend upon the mere sensations and statements of the patient, but which is attended with, and in fact consists of, distinct physical alterations which can be seen by the physician. Double optic neuritis is in fact more than a mere symptom; it is in the strictest sense of the term a physical sign — an objective and visible indication of disease which is infinitely more important than any mere subjective sensation, such as headache or dimness of vision, or even complete blindness.

Secondly, Because it is present in the great majority of cases of intracranial tumour, *at some period or other of their course.* Statistical observations seem to show that double optic neuritis is probably present in eighty per cent. of the cases of intracranial tumour, at some period or other of the course. The proportion of cases in which double optic neuritis is present, when the patient first comes under the observation of the physician—in other words, at the time when he is first asked to give an opinion as to the nature of the case—is undoubtedly much less than eighty per cent. Nevertheless, double optic neuritis is so frequently present, even when the patient first comes under observation, that it is generally recognised as one of the most frequent symptoms (and when present, for the reasons which are just being stated, the most important symptom) of the disease.

Thirdly, Unlike headache, vomiting, and vertigo, double optic neuritis is a physical sign which is not commonly produced by other conditions; or, to state the matter more accurately, an intracranial tumour is by far the most common condition with which double optic neuritis is associated. If we were to take 100 cases of Bright's disease, of lead poisoning, of meningitis, or of anæmia, the proportion of cases in which double optic neuritis would be present—even granting that we could observe the case from first to last—would be infinitely less than eighty per cent. This statement is not, however, applicable to those cases of lead poisoning, which are likely to be mistaken for an intracranial tumour. In the form of lead poisoning (lead encephalopathy, as it is termed), in which head symptoms are prominent, double optic neuritis is in my experience very generally present. I am disposed to think, that in acute lead encephalopathy, double optic neuritis is quite as frequently present as in cases of cerebral tumour.

The "*localising*" or "*focal*" symptoms which may result from the presence of an intracranial tumour are very numerous and very important. They result from the pressure of the tumour (or the meningitis which surrounds it), upon some individual part of the brain, or upon some special portion of the intracranial contents or of the structures forming the boundaries of the cranial cavity (nerve trunks at the base of the brain, blood vessels, membranes, bones forming the cranial wall, &c.).

The pressure of a new growth upon nerve tissue, or indeed upon any tissue, is attended with one or other of two results—destruction or irritation. In many cases, both results are indeed produced. In many cases, the nerve tissue is irritated before it is destroyed. It not unfrequently happens that irritation and destruction are simultaneously produced—that while one portion of the nerve tissue is destroyed, or so pressed upon that its function is arrested or

inhibited, another and adjacent part is irritated, and its functional activity is excited. Localised paralysis and spasms, localised derangements of sensation, such, for instance, as hemianopsia, and the various speech derangements, which are grouped together under the common term aphasia, are some of the chief localising symptoms of a positive kind. I say of a positive kind, for it is of the greatest importance to remember that, in trying to determine in what particular part of the cranial cavity a tumour is situated, we have to be guided not only by the positive facts, but in some cases quite as much by the negative indications which are present. Given, for instance, the presence of symptoms and signs indicative of the presence of an intracranial tumour in some part of the cranial cavity, the negative fact that there is neither paralysis nor spasm, enables us to say that the tumour is, in all probability, not situated in the motor area. I say in all probability, for, in rare cases, very remarkable exceptions to this general rule are met with. I have myself recorded a case, in which the greater part of the motor area in the left hemisphere of the brain was destroyed by a large sarcomatous tumour, and in which there was no paralysis. The case is of so much interest and importance that I make no apology for reprinting it in full. (See page 278.)

I do not propose on the present occasion to describe in detail the exact character of the localising or focal symptoms which may result from the presence of an intracranial tumour in different parts of the cranial cavity. In order to do so satisfactorily, I should have to consider the whole subject of cerebral localisation—a subject of the greatest interest and importance, but one so vast that it would be impossible to treat it satisfactorily in the space at my disposal.

I should like, however, to illustrate the value of the localising or focal symptoms by means of one symptom, viz., hemiplegia.

The ordinary, common, form of hemiplegia—in which the face, arm, and leg on one side of the body are paralysed—is, *par excellence*, the typical form of cerebral (as distinct from spinal, or peripheral) paralysis.

In the great majority of cases in which the face, arm, and leg on one, and the same, side of the body are paralysed, the lesion is situated in the hemisphere of the brain on the side opposite to the paralysis; in other words, the lesion involves the intra-cerebral motor mechanism (motor centres, or conducting fibres of the pyramidal tract) above the crus cerebri. (See 5, fig. 90.)

The ordinary form of hemiplegia, to which I am at present referring, may, of course, be due to a variety of different lesions, amongst which hæmorrhage in the neighbourhood of the internal capsule and embolism or thrombosis of the middle cerebral artery are by far the most common.

A cerebral tumour is one of the lesions which may produce the ordinary, typical form of hemiplegia.

It is rarely, however, that the hemiplegia, which is due to the presence of a cerebral tumour, in one (the opposite) cerebral hemisphere is so complete and so extensive in its distribution as the hemiplegia which is due to a well marked cerebral hæmorrhage, or to complete plugging of the middle cerebral artery I refer to those cases in which the hemiplegia is permanent; in other words, to those cases in which the paralysis is due to direct pressure upon, or destruction of, the motor nerve mechanism above the crus cerebri.

The temporary form of hemiplegia, which we term post epileptic, which is not uncommon in the course of syphilitic gummata and other "coarse" lesions (tumours, etc.), and which follows an attack of one-sided (unilateral) epileptiform convulsions or Jacksonian epilepsy, may for the brief period that it lasts (the few days of its duration), be quite as complete in degree and as extensive in its distribution as the hemiplegia which is due to hæmorrhage or embolism.

But the hemiplegia which a tumour produces by direct pressure upon, or destruction of, the motor nerve mechanism, above the crus cerebri, rarely, at its commencement at all events, involves all the muscles on the opposite side of the body—all those muscles which are paralysed as the result of a hæmorrhage or

embolism (*e.g.*, the muscles of the face, arm, and leg).

The hemiplegia which is due to the presence of a tumour in the opposite cerebral hemisphere is in most cases incomplete, at its commencement, both as regards the extent and the degree of the paralysis. The loss of muscular power may, for a time, involve the face only, or the arm only, or the leg only; and the paralysis of the part which is first involved (face, arm, or leg), is usually for a time imperfect; to use a popular, but I hold an erroneous, expression, it is a paresis rather than a paralysis. It is only after a time, and as the tumour increases in size, and involves more and more of the motor mechanism (cortical centres or conducting fibres), that the face, arm, and leg are also involved (*i.e.*, that the hemiplegia becomes complete, as regards the extent and distribution of the paralysis); or, to put the matter in another way, a tumour of the cerebral hemisphere, in many cases, first produces a monoplegia, which in the course of time may become a hemiplegia.

This slow and gradual mode of development of the paralysis is very characteristic, and of great diagnostic importance. It is highly suggestive of the presence of a slow-growing and gradually progressive lesion, such as a tumour.

I do not mean to say that the hemiplegia which is due to the presence of a tumour in one cerebral hemisphere is always produced in this slow and gradual manner. Cases of cerebral tumour are occasionally met with in which permanent paralysis of the face, arm, and leg (exactly resembling, both in the extent and in the degree of the paralysis, the hemiplegia due to hæmorrhage or embolism) is suddenly developed. Let me give an illustration. Hæmorrhagic extravasations are very frequently due to the rupture of the thin walled and dilated vessels, which are such conspicuous features in many gliomatous tumours. If the hæmorrhage is extensive, or if the glioma is situated in the neighbourhood of the motor portion of the internal capsule, paralysis of the face, arm, and leg, on the opposite side, may be suddenly developed. But cases of this kind are comparatively rare. The slow and gradual mode of development of the paralysis is much more common.

In most cases in which a tumour of the hemisphere produces hemiplegia (I refer to the permanent form of hemiplegia which is due to destruction of, or direct pressure upon, the motor nerve mechanism above the crus cerebri), the tumour is situated, either on the surface of the brain, in the cortex, or in the white matter immediately below the cortex (the subcortical portion of the centrum ovale).

Now a tumour, in these situations, which produces paralysis of the face, arm, and leg, must necessarily be of large size. In order to produce paralysis of the face, arm, and leg (permanent paralysis, not merely temporary or functional paralysis), it must necessarily involve the whole of the motor area (the cortical centres for the face, arm, and leg), or the fibres in the subjacent white matter which proceed from those centres.

A small tumour, situated in the deeper parts of the centrum ovale, or in the immediate neighbourhood of the motor part of the internal capsule, *i.e.*, which involves the fibres of the pyramidal tract after they have conveyed and become collected together in a comparatively narrow area, may, of course, produce paralysis of the face, arm, and leg, on the opposite side. But, as a matter of fact, tumours are more frequently situated on or in the cortex, or in the subcortical white matter, than in the basal ganglia or deeper parts of the centrum ovale. Hence it is that the hemiplegia, which is due to the presence of a tumour in the cerebral hemisphere, is seldom so extensive (at its commencement at all events), as the hemiplegia which is due to a hæmorrhage in the lenticulo-striate region, or to embolic plugging of the middle cerebral artery.

The manner, then, in which a hemiplegia is developed in cases of cerebral tumour, is, in many cases, of great diagnostic value (*i.e.*, is indicative of a tumour).

Further, the fact that the paralysis is, in many cases of tumour of the hemisphere, limited, at its commencement, to the face, arm, or leg (in accordance with the position of the tumour, and the function of the cortical

centres or conducted fibres which are first implicated by it), is of distinct localising value.

Further, by observing the way in which the paralysis extends (the order in which the face, arm, and leg become paralysed, or the "march" of the paralysis, as it may be termed), we are able, in some cases, to form an (approximate) opinion as to the direction in which the tumour is extending; for, as has been already pointed out, the progressive increase of the paralysis is, in many cases, due to the fact, that motor centres or conducting fibres, which at the commencement of the case were uninvolved, have gradually become encroached upon, or invaded by, the new growth.

Granting, then, that the ordinary, typical form of hemiplegia is associated with the general symptoms (headache, vomiting, and optic neuritis), indicative of an intracranial tumour, the physician is justified in concluding that the tumour is, in all probability, situated in the hemisphere of the brain, on the opposite side to the paralysis.

This conclusion is, however, only justifiable when the hemiplegia is of the ordinary typical variety (*i.e.*, when the paralysis involves the face, arm, and leg, on one and the same side), and when the hemiplegia is the only localising symptom—or, to put the matter more accurately, when no other localising symptoms are present which indicate that the tumour is situated at the base of the brain, in the pons Varolii, medulla oblongata, or cerebellum.

It is important to remember, that tumours, which are situated at the base of the brain, in the pons Varolii, medulla oblongata, or cerebellum, and which press upon or involve the fibres of the pyramidal (motor tract), in or below the crus cerebri, may give rise to hemiplegia.

In most cases of this kind, the hemiplegia is not the ordinary typical form. Tumours which press upon or involve the crus cerebri on one side, or which are limited to one lateral half of the pons Varolii or medulla oblongata, usually produce irregular or alternate hemiplegia—"crossed" hemiplegia, as it is sometimes, but very badly, named.

Several different varieties of alternate or irregular hemiplegia are met with. The essential facts to remember, in connection with all these irregular forms of hemiplegia, are:—

Firstly, That the lesion (tumour) involves the main pyramidal tract above its decussation (at the lower end of the medulla), and, consequently, produces paralysis of the arm and leg, and it may also be of the face, on the opposite side of the body.

Whether the face is paralysed, as well as the arm and leg, on the opposite side to the lesion (tumour), depends, of course, upon the position of the lesion, *i.e.*, whether it is situated above or below the decussation of the fibres of the facial nerve in the pons Varolii.

A lesion in the upper part of the pons Varolii, which involves the main pyramidal tract on one side, will involve the facial nerve fibres above their point of decussation, and will, consequently, produce paralysis of the face muscles (as well as paralysis of the muscles of the arm and leg), on the opposite side of the body. In other words, the face, arm, and leg, on the opposite side to the lesion, will be paralysed. So far as the face, arm, and leg are concerned, the hemiplegia which results from a lesion of this description, differs in no way from the hemiplegia which is due to a lesion above the crus cerebri, *i.e.*, from a lesion which involves the motor portion of the internal capsule.

A lesion in the lower part of the pons Varolii or in the medulla oblongata, which involves the main pyramidal (motor) tract on one side, will produce paralysis of the arm and leg on the opposite side. A lesion in these situations may not produce any facial paralysis, for it may be situated below the point of emergence of the fibres of the facial nerve (below the superficial origin of the facial nerve). If the fibres of the facial nerve are involved by such lesions, (tumours situated in one lateral half of the lower end of the pons Varolii or in the medulla oblongata), the paralysis of the face will be on the same side as the lesion; for the fibres of the facial nerve will be involved *below* their decussation in the pons Varolii.

These facts are diagrammatically represented in figs. 90, 91, and 92.

Secondly, That the lesion (tumour) involves the fibres of some one or more of the cranial nerves *below* their point of decussation, *after* they have crossed the middle line, as they are passing through their nerve nuclei, or as they are emerging at their points of superficial origin from the pons Varolii or medulla oblongata.

The more important of the irregular or alternate forms of hemiplegia are as follows:—

1. Paralysis of the face, arm, and leg on one and the same side (the side opposite to the lesion), and paralysis of the muscles supplied by the third nerve on the same side as the lesion.

This form of alternate hemiplegia is usually due to a lesion (say a tumour at the base of the brain which presses upon the crus cerebri).

Fig. 89, which is copied from a photograph, is an exact representation of a case of this kind. The patient, a child aged eighteen months, came under my observation some two or three years ago. She suffered from vomiting, but there was little headache, and no optic neuritis. The fontanelle was unclosed. I have in some other cases of intracranial tumour, in which the fontanelle was unclosed, noted the absence of optic neuritis. There was very marked paralysis of the muscles supplied by the third nerve, on the left side. The ptosis is well shown in the drawing. The arm and leg on the right side were also paralysed, but not completely so. The hand, which was most affected, was rigid, and the fingers spasmodically contracted into the palm. I was unable to satisfy myself as to the presence of any distinct paralysis of the facial muscles. The paralysis of the muscles supplied by the third nerve, on the left, and of the arm and leg, on the right side had been slowly and gradually developed.

I diagnosed a scrofulous tumour at the base of the brain, pressing upon the left crus cerebri.

Under appropriate feeding, and the adminis-

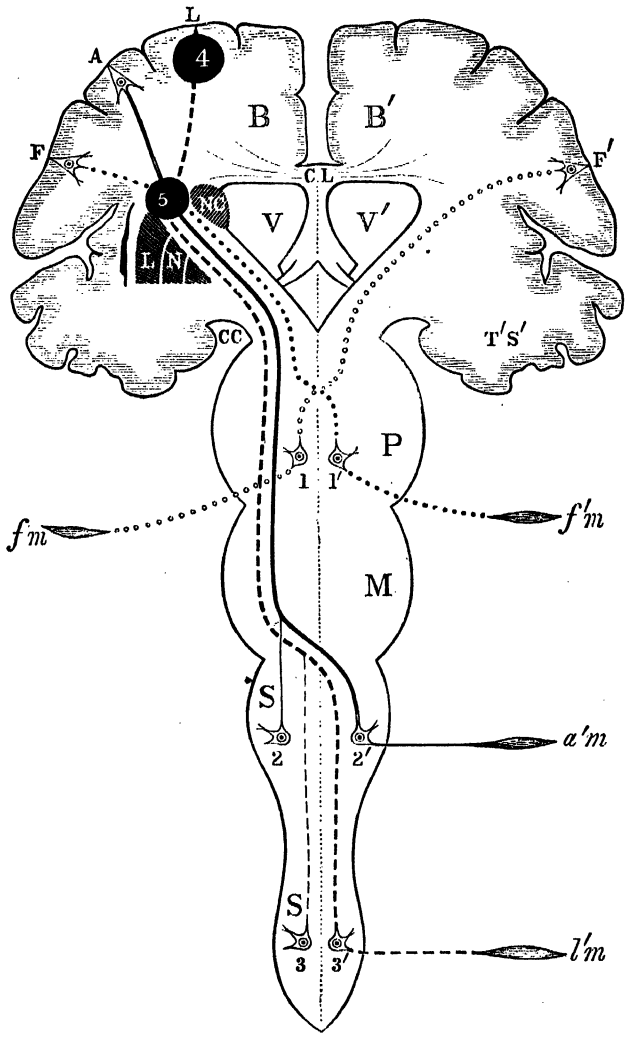


FIG. 90.—Diagram showing the arrangement of the motor tracts for both sides of the face, the left arm, and the left leg; and the distribution of the paralysis produced by lesions in different parts of the cerebrum.

B, the right, and *B′*, the left cerebral hemisphere; *P*, the pons Varolii; *M*, the medulla oblongata; *S*, *S*, the spinal cord; *V′ V′*, the lateral ventricles; *NC*, the nucleus caudatus; *L N*, the lenticular nucleus; *C C*, the crus cerebri; *T′ S′*, left temporo-sphenoidal lobe; *F*, *A*, *L*, cortical centres for the face, arm, the left lower extremity; *fm* and *f′m*, right and left facial muscles; *a′m*, muscles of left upper, and *l′m*, muscles of left lower limb; 4, localised lesion in the cortex, producing paralysis of the opposite leg; 5, lesion of the pyramidal tract as it enters t..e internal capsule, producing hemiplegia on the opposite side of the body (paralysis of the face, arm, and leg).

3, 3′, rig

FIG. 89.—Case of crossed hemiplegia (ptosis of the left eye, and paralysis with spasm of the right arm and leg), the result of a scrofulous tumour in the neighbourhood of the left crus cerebri.

tration of iodide of potassium and the syrup of the iodide of iron, the paralysis slowly and gradually disappeared; and in the course of eight or nine months the child grew fat, and seemed to be perfectly well. I regret very much that I have no photograph of the patient at this period of the case.

Some ten months after the patient first came under my observation, the vomiting again returned, headache was complained of, and ultimately symptoms indicative of tubercular meningitis developed.

On my recommendation the child was admitted to the Sick Children's Hospital, where, in the course of a short time, she died.

At the post mortem examination, a scrofulous tumour was, I understand, found at the base of the brain in the region of the left crus cerebri. Another scrofulous tumour was situated in the cerebellum; and there was also some tubercular meningitis.

The case is of considerable interest, because of the very remarkable improvement which took place under treatment

2. Paralysis of the arm and leg on the opposite, and of the face on the same side, as the lesion.

This form of alternate hemiplegia is usually due to a lesion in the lower part of one lateral half of the pons Varolii (see 7, fig. 91).

3. Paralysis of the arm and leg on the opposite, and of the muscles supplied by the seventh (facial), and by the sixth (external rectus) nerve on the same side.

This form of irregular or alternate hemiplegia is usually also due to a lesion in the lower part of one lateral half of the pons Varolii.

4. Paralysis of the arm and leg, on the opposite, and of the facial muscles on both sides.

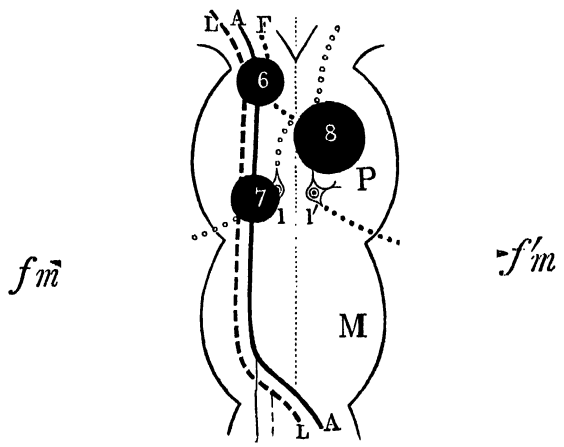


FIG 91.—Diagram showing the arrangement of the motor tracts fc both sides of the face, the left arm, and the left leg in the pons Varoli and the distribution of the paralysis produced by lesions of differer parts of it.

6, lesion in the upper part of the right side of the pons Varolii, produc ing paralysis of the face, arm, and leg on the opposite (left) side; 7, lesio in the lower part of the right side of the pons Varolii, producing paraly sis of the face on the same, and of the arm and leg on the opposite side 8, lesion in the middle of the left side of the pons Varolii, producin paralysis of both sides of the face, and of the arm and leg on the opposit side,—the conducting tracts passing to the right arm and leg are nc shown in the left side of the pons, but they would obviously be inte

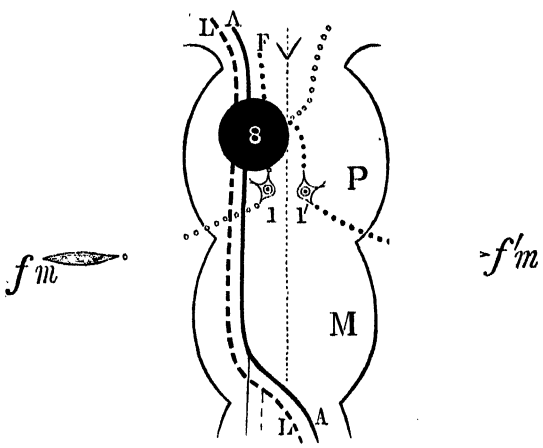


FIG. 92.—Diagram shewing a lesion in the right half of the pons Varolii, causing paralysis of both sides of the face, and of the arm and leg on the opposite left side.

The letters *F*, *A*, *L*, point to the motor tracts for the left side of the face, arm, and leg, as they enter the right side of the pons Varolii (*P*) in their passage from the right hemisphere of the brain. (Compare with Fig. 90.)

A lesion in the position of 8 (middle of the right side of the pons) may cause paralysis of the opposite arm and leg, and of both sides of the face (*fm* and *f'm*), by intercepting the motor fibres proceeding to the left side of the face (*f'm*), before they have decussated, and those passing to the right side of the face (*fm*) after they have decussated.

This form of alternate hemiplegia may be produced by a lesion in the pons Varolii, which implicates the fibres of the pyramidal tract for the arm and leg on the opposite side, and the fibres of the facial nerves on both sides, *i.e.*, the fibres passing to one (the opposite) side of the face before their point of decussation, and the fibres of the facial nerve on the other (the same) side, after their decussation (See 8, fig. 92).

5. Paralysis of the arm and leg, on the opposite, and of the tongue on the same side as the lesion.

This form of alternate hemiplegia may be due to a tumour in one lateral half of the medulla oblongata, which involves the fibres for the arm and leg, on the opposite side, before (above), and the fibres of the hypoglossal, on the same side, after their decussation.

It is obvious from the description which has just been given, that, by observing the exact distribution of the paralysis in the different varieties of alternate hemiplegia, very valuable information may in some cases be obtained as to the exact position of an intracranial tumour —granting, of course, that the lesion which produces the hemiplegia is (shown by the other symptoms which are present to be) a tumour. The mere fact that there is alternate hemiplegia does not of course warrant a diagnosis of tumour. The fact that an alternate hemiplegia is slowly and gradually developed is, however, very suggestive of tumour.

II.—Optic Neuritis.

Inflammation of the optic disc is a condition of the greatest diagnostic value, for its presence enables the physician in some cases to determine that the symptoms are due, not merely to functional disturbances, but to the presence of serious organic disease. As I have already pointed out, optic neuritis is a condition which does not depend upon the mere statements and sensations of the patient, but which is attended with distinct physical alterations which can be seen by the physician.

The degree of change varies from slight œdema, which just obscures as a greyish haze, the normal sharp outline of the disc, and renders the vessels, more especially the arteries, somewhat indistinct as they pass over the edge of the disc, to the most intense inflammatory swelling, which completely obscures the outlines of the disc, involves the adjacent portions of the retina, completely hides the lamina cribrosa, and extensively covers up the blood vessels, more especially the arteries. In well marked cases, hæmorrhagic extravasations are very often present both upon the swollen disc and the surrounding retina. In consequence of the pressure to which they are subjected, the arteries are usually diminished in size, and the veins enlarged and tortuous. The disc often has a mossy, velvety, striated, and in some cases, purplish-grey appearance.

In consequence of the extension of the swelling over the adjacent retina, the disc looks larger than normal.

Optic neuritis may entirely subside, leaving vision not at all or but little affected—a result which is undoubtedly aided by energetic treatment (iodide of potassium in full doses and mercury), but in many cases, permanent damage to the optic nerve results; and, when the neuritis subsides, more or less atrophy, with more or less dimness of vision, remains.

In old standing cases of well marked optic neuritis, vision is usually more or less affected; and in some cases there is complete blindness. But it is of the utmost importance (both for the purposes of diagnosis, and also for the prevention of permanent blindness by a vigorous plan of treatment), to remember, that vision (both the acuity of vision and the extent of the visual fields for white and for colours) may be absolutely normal, even when the most marked double optic neuritis can be seen with the ophthalmoscope.

One of the most striking cases of the kind which has come under my own observation was that of a man aged twenty-three, who was sent to me a short time ago by Dr Berry. The optic neuritis was double, and extremely well marked; it had been observed by Dr Berry for several weeks, before the case came under my observation. The exact causation was obscure; the patient had apparently had an attack of meningitis, characterised by headache, vomiting, and high temperature; there was some double vision, but there were no symptoms which enabled me to positively diagnose the presence of an intracranial tumour—a condition which naturally suggested itself—and a guarded prognosis was given. The optic neuritis lasted for

many weeks after the patient first came under my notice, but ultimately completely disappeared under the continued administration of large doses of iodide of potassium, and the patient has since been in every respect well. The acuity and field of vision, which were regularly examined, were never in the least degree impaired; both at the height of the condition, and when the inflammatory changes had completely disappeared, they remained absolutely normal. The patient had not had syphilis.

I have reported several other cases of intracranial tumour, one of which is represented in figs. 94 and 95, in which well marked double optic neuritis was associated with good or perfect central vision—a fact which was long ago insisted upon by Dr Hughlings Jackson, and which is now widely recognised by all competent authorities.

Optic neuritis may be single or double. Unilateral optic neuritis is, in the great majority of cases, due to local disease in the affected eye or orbit, and comes under the notice of the surgeon much more frequently than of the physician. One-sided optic neuritis is so rarely due to intracranial or general disease, that its presence is strongly suggestive of local disease in the cavity of the orbit, or at all events of a lesion of the trunk of the optic nerve in front of the chiasma. A tumour at the base of the brain growing from the margins of the orbital fissure or optic foramen, may, however, in its earlier stages implicate one optic nerve only, and produce one-sided optic neuritis. In many of these cases, the optic nerve on the other side subsequently becomes affected, and the optic neuritis becomes double. In some rare cases of intracranial tumour in which the trunk of the optic nerve is not directly implicated, the neuritis has been entirely confined to one eye, throughout the whole course of the case. Dr Hughlings Jackson thinks that in cases of this description, the disc on the opposite side to the brain lesion is more frequently affected than that on the same side; but whether this is more than a mere accidental coincidence, and whether it is a point of real importance is perhaps doubtful. The number of cases of intracranial tumour, in which the optic neuritis has been unilateral, is, in my opinion, too small to allow of any definite generalisation being made.

In the vast majority of the cases of optic neuritis, which are associated with medical affections, and which, therefore, come under the care of the physician, the condition is bilateral; though it very frequently happens that the inflammatory changes are more marked in one eye than the other.

The condition with which double optic neuritis is most frequently associated is undoubtedly an intracranial tumour, but it also occurs in connection with meningitis (especially basilar meningitis, in which it is common); abscess of the brain; the very rare condition, chronic diffuse cerebral atrophy or diffuse chronic cerebritis; Bright's disease (more especially the cirrhotic form of kidney disease); lead poisoning; idiopathic and other profound forms of anæmia (chlorosis leucocythæmia, etc.); suddenly arrested menstruation; sudden distension of the sheath of the nerve with blood, and probably also (though much more rarely) with some other conditions.

The exact manner in which some of these conditions produce optic neuritis is not always clear. In some cases, it is undoubtedly due to descending neuritis; in others, it appears to result, in part at least, from distension of the sheath of the nerve, with blood or other fluids, and from the inflammatory changes which result therefrom; in some cases, it is probably the result, as Leber and Deutschmann have suggested, of irritating or infective particles being carried by means of the subarachnoid fluid from the cavity of the brain to the vaginal sheaths surrounding the nerve. I have elsewhere summed up my views as to the causation of the double optic neuritis which is associated with intracranial tumour as follows:—"It must be allowed that in some cases it is due to a "descending" inflammation, but this does not appear to me to be the usual or most common cause of the condition. The pressure-irritation theory is, in my opinion, the most likely explanation in the majority of cases. Increased intracranial pressure seems to me an important

though perhaps a secondary and favouring cause of the condition. The essential and primary cause is probably in many cases, as Leber and Deutschmann have suggested, the presence of an irritant in the cerebro-spinal fluid. But even if we accept the theory which has been advanced by Leber and Deutschmann, it must, I think, be allowed that increased intracranial pressure is an important factor in the production of the optic neuritis, which is associated with intracranial tumours. The increased pressure within the cranium forces the arachnoid fluid into the subvaginal space, with the result that the irritant contained in the subarachnoid fluid produces inflammatory changes in the vaginal space, optic nerve, and optic papilla. Before the Leber-Deutschmann theory can be definitely adopted, the presence of an irritant, and its exact nature, require, I think, further demonstration. The weight of evidence seems to me strongly opposed to the vaso-motor view."

The differential diagnosis of the cause of double optic neuritis (*i.e.*, the exact condition with which the optic neuritis is associated) is often a difficult matter, and in many cases can only be determined by a judicial survey of all the symptoms, physical signs, and associated pathological conditions, together with an accurate history of their duration, exact mode of origin, and progress, up to the time when the patient comes under the notice of the physician. It is impossible to attempt to discuss the matter here; the reader who wishes further information on the point will find full details in my work on Intracranial Tumours (Chapter VII.).

III.—The Treatment of Typhoid Fever.

(*Continued from page* 263.)

In a case of typhoid fever, the nurse has to attend to the following points:—

In the first place, she has to see that the surroundings of the patient—more especially the temperature and ventilation of the sick-room—are properly attended to. During the small hours of the night, when the temperature falls, she must take care that the fire is kept good; for, as I have already pointed out, it is desirable that the temperature of the sick-room should, so far as possible, be maintained at a fixed point (at, or a little below, 60° Fahr.).

In the second place, the nurse has to see that the patient is supplied, at regular stated intervals, with a sufficient amount of satisfactory nourishment. In typhoid fever, the feeding of the patient constitutes one of the most important points in the management and treatment of the case.

In the third place, the nurse must see that the medicine and stimulants, which are prescribed by the doctor, are administered to the patient at the proper times, and in the proper quantities.

In the fourth place, she must see that the patient is kept clean, and that the evacuations, which in some bad cases of typhoid are discharged involuntarily, are not allowed to lie in contact with the patient. She must also see that the discharges from the bowels are disinfected immediately they are evacuated, and that all soiled linen is immediately removed, and placed in some disinfecting fluid. The satisfactory disinfection of the intestinal evacuations, is a matter of great practical importance; it is a point on which the safety of the other inmates of the house, and possibly of the other inhabitants of the town or district (in which the case is situated), may depend. It is unnecessary to say, that personal cleanliness on the part of the nurse herself, is for many reasons of the greatest importance. It is especially important, so far as her own safety is concerned, that she should take care to keep her hands scrupulously clean, to wash and disinfect them every time they become in the least degree soiled by the evacuations of the patient. There can, in my opinion, be no question, that in many of the cases in which a nurse takes typhoid from a patient, the disease is communicated in consequence of some particle of the intestinal evacuations being

allowed to remain on her hands, or about her person. It is easy to see that the typhoid poison, which is contained in the matter evacuated from the intestine, may without difficulty find its way from the hands of the nurse to her mouth, and so into the alimentary canal—the channel through which the poison is usually introduced into the system.

In the fifth place, the nurse should frequently examine the patient's back, and take measures to guard against the production of a bed sore.

In the sixth place, she must take the temperature at regular stated intervals, and keep a written record of the progress and course of the case between the visits of the doctor.

The nurse should mark down on a piece of paper, the amount and kind of food, and the amount and kind of stimulant which are administered, the exact time at which these articles are given being of course stated. Further, she should note the nature of the medicine which she gives to the patient, the dose, and the time at which it is administered. She should also keep a record of the number of times the bowels are evacuated, and of the character of the evacuations. She must, of course, take the temperature at regular stated intervals, and record each observation on a chart. Lastly, she should note down any other points in connection with the case which seem to her to be of importance. If, for example, there is much cough; if the patient is very restless or delirious; if he makes any special complaints; if he becomes cold or collapsed, the fact should be noted down in writing.

By the help of such a written record, the doctor gains much valuable information, not only as to the way in which the case is progressing, but also as to the manner in which the nurse is carrying out her duties.

In the seventh place, the nurse must see that the patient, while in a state of delirium, does not get out of bed, or attempt to go out of the room, or jump out of the window.

Lastly, the nurse should be able to recognise the occurrence of any new or serious symptoms, and be prepared, should the patient seem suddenly to be worse, to send off immediately for the doctor.

It is essential during the early stages of the attack, when the patient is acutely conscious, complaining of violent headache, thirst, great restlessness, and general malaise and depression, that the nurse should endeavour by every means in her power to relieve his sufferings, and to get him to take a hopeful and cheerful view of his condition. Again, after the acute stage of the disease is past, during the earlier stages of defervescence, when the delirium has passed off, and the patient has again become conscious (if he was unconscious during the acute stage of the disease—and in severe cases there is usually more or less delirium and unconsciousness), it is very important that the nurse should be kind, attentive, and sympathetic. During the earlier periods of convalescence, the patient is often in a very miserable and dejected condition—restless and acutely conscious of his feebleness and suffering.

A thoroughly good and satisfactory nurse should consequently be an active, intelligent, cheerful, well meaning and thoroughly sympathetic woman, who takes a real personal interest in her patients. It is a very unfortunate thing for the patient when he gets a nurse who performs her duties in a merely routine manner—because she has to do them—and who takes no active and personal interest in his sufferings.

A nurse who is required to take charge of a case of typhoid, should be possessed of a considerable amount of bodily strength; at all events, she should not be a weak and delicate woman, for the strain and fatigue which necessarily attend the nursing and management of a bad case are very considerable. When there are two nurses—one for the night and another for the day—(and as I have already pointed out, this is eminently desirable), a great deal of the strain is removed.

It is essential, of course, that the nurse should be a sober woman. It may perhaps appear unnecessary to make this stipulation at the present day, when the character of our nurses, both in private and hospital practice, is so very

different in this and many other respects from what it was a few years ago. But even now, it is necessary to keep one's eyes open to the failings of nurses as regards alcohol. A nurse is placed in a position of great delicacy and trust. If she is addicted to stimulants, she can easily gratify her wish. The stimulant is there, she has only to take it, there is no one to prevent her; she has every opportunity, should she feel inclined to do so, to take the stimulants which are ordered for the patient. It is very essential, therefore, that the medical man should keep his eyes open with regard to this point; and indeed, with regard to the whole way in which the nurse performs her duties. It is a good plan to take the nurse every now and again unawares—to vary from time to time the hour of one's visit—to look in and see the patient every now and again when the nurse does not expect you. If you always make your visit at the same hour, a nurse who is careless and neglecting her duty between times, may have everything in apple-pie order when the doctor is expected; at the time of your visit everything may be spick and span. It is only by varying the hour of the visit, and taking the nurse unawares, that we can see whether things are going on satisfactorily between times or not. It is important, too, to remember that the physician should not be content with asking the nurse whether the patient is clean, whether the back is right, whether the bladder has been satisfactorily evacuated. At every visit the medical attendant should investigate all these points for himself. By visiting the patient when you are not expected, and by examining all these points for yourself (turning down the bedclothes, seeing that the patient is clean and dry, looking at the back, palpating and percussing the bladder, &c.), you very soon come to find out the sort of nurse you are dealing with—and what is quite as important, the nurse very soon comes to realise the sort of doctor she is dealing with. A little unspoken, but very easily understood, explanation of this kind between the nurse and the doctor, is eminently advantageous to the welfare of the patient.

As I have already pointed out, it is desirable that a nurse who is to take charge of a case of typhoid fever should have had some previous experience of the disease; it is very desirable that she should know exactly what to do, what to expect, and that she should be able to recognise any serious symptoms as soon as they arise, in order that she may immediately communicate with the doctor.

Provided that the nurse is a reliable, judicious, and thoroughly trustworthy person, it is desirable that she should have full charge of the sick-room, in the absence of the doctor. The friends and relatives of the patient must, of course, be made to understand that the nurse has, under the orders of the medical attendant, full direction of the patient; they must not be allowed to unduly interfere with her, or to dictate to her as to what is and what is not to be done. The nurse should have full charge of the sick-room in the absence of the doctor; she is responsible to the doctor, and to him alone.

Although the nurse should be (provided that she is a thoroughly reliable and capable person) in full charge of the sick-room, it is very desirable that some near relative of the patient should be every now and again in and out of the room, and should keep an eye on the way in which she is performing her duties. If the relatives have any suggestion, or any complaint to make, as to the nursing or as to the way in which the nurse is conducting herself, they should make it to the doctor. It is his duty to investigate the matter, and to adjudicate upon it.

It is, of course, very desirable that the nurse should understand, that she is merely the nurse and not the doctor. Some nurses are apt to think that they know quite as much as the doctor, and to assume duties which should only be assumed by the medical man. A clever and experienced nurse is very apt to think, that she knows more than a young doctor (and in some cases she is doubtless right), and to presume accordingly. It is very desirable that the medical man should from the first make the nurse clearly understand, by his whole bearing, manner, and address, that he is the

master—that she is the nurse and not the doctor. Whenever a nurse shows any tendency to presume or to interfere with matters which belong to the province of the doctors, she should be immediately and sharply pulled up. If the nurse is a really clever and able woman, as well as a good experienced nurse, she very soon appreciates the true character of the doctor. She very soon sees whether he is a man who is likely to be imposed upon, or to put up with any nonsense; and she takes care to regulate her conduct accordingly.

With these preliminary remarks on the nursing of the patient, I now pass to consider the third point in the treatment of a case of typhoid, viz., the *feeding of the patient.*

The administration of a sufficient amount of suitable food is one of the most important—perhaps the most important—point in the management and treatment of a case of typhoid fever.

In considering the feeding of a typhoid fever patient, it is essential to remember;—

Firstly, That we are dealing with a febrile disease of several weeks' duration; that the main object of our treatment is to tide the patient over the attack; and that one most important means of enabling the patient to battle with the disease, is to sustain his strength by the administration of a sufficient supply of nourishment.

Secondly, That the local lesion consists in an inflamed and ulcerated condition of the intestine; and that it is essential to avoid giving anything (whether food or medicine) which is likely to increase the diarrhœa, and irritate the inflamed and ulcerated gut.

In typhoid fever, as in all febrile and inflammatory affections, liquid food should alone be given during the active stages of the attack. Milk should, of course, constitute the main article of food for a fever patient. Beef tea, chicken or veal broth, and egg whisked up with milk, or a little wine and milk, or some of the various extracts of meat, such as Valentine's beef juice, peptonised chicken jelly, &c., may also be given. But milk and milk foods should undoubtedly be the chief articles of the dietary. Unfortunately some people possess a peculiar idiosyncrasy, which renders them unable to take and digest milk. For such cases, peptonised milk may be tried. Benger's food is in many cases of this kind eminently serviceable. I may here say, that in many cases of typhoid—many of the cases in which there is no natural inability to take and digest milk—the peptonised milk is eminently beneficial. It not unfrequently happens, that if a typhoid fever patient is fed exclusively on milk, or if large quantities of milk are given to him, that he gets more milk than he can satisfactorily digest. The result is, that undigested curds pass into the intestine, irritate the inflamed and ulcerated gut, and increase the diarrhœa. Sir William Jenner has pointed out the danger of giving too much milk in typhoid, and has directed attention to the fact, that when the diarrhœa is excessive, the stools should be examined, with the object of determining whether undigested curds of milk are passing into the intestine, and so causing the diarrhœa. Some persons do not like the taste of peptonised milk, but it is seldom that patients who are suffering from typhoid fever object to it; the tongue, it must be remembered, in typhoid is (at the height of the disease at all events) coated and dry; the sense of taste is much impaired; and most patients, so far as my experience enables me to judge, take the peptonised milk quite readily.

If beef tea, chicken broth, veal broth, are given as substitutes for milk, or along with milk, in cases of typhoid fever, their effect must be carefully watched. If they increase the diarrhœa, they should be given in smaller quantity, or omitted altogether from the dietary.

It is essential to remember, that although the appetite is usually completely lost in cases of typhoid, the patient is very thirsty. He craves for water. Milk does not, as a rule, quench thirst. This craving on the part of the patient for water, is one of nature's indications, which should be carefully attended to. In any long-continued febrile disease, the heated body requires water, hence the thirst. Water is

demanded by the overheated tissues. The administration of water to a fever patient helps, amongst other things, the action of the kidney, and favours the evacuation of urea and other excrementitious products, which, if retained in the blood, are apt, as we have seen, to exert a very deleterious effect upon the nerve centres. Water should therefore be freely given to fever patients, with the object of allaying thirst, supplying the demand of the over-heated tissues for fluid, and as a diuretic. A large quantity must not, of course, be given all at once, but the patient should be encouraged to take small quantities at frequent intervals. If the patient likes it, he may every now and again suck a small piece of ice. With the object of quenching thirst, a small quantity of acid may be added to the water. Citric acid, or a little lemon juice, or a few drops of some mineral acid, such as hydrochloric acid, may be given. In the earlier stages of the attack, when the headache is usually such a troublesome symptom, hydrobromic acid is, I believe, often beneficial, both as a nervine sedative and as a thirst quencher.

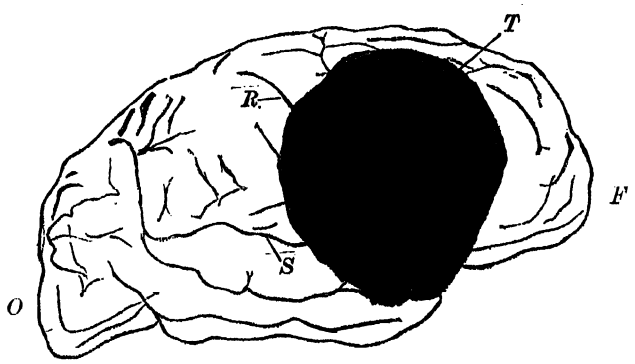


FIG. 93.—Diagrammatic outline of the surface of the right hemisphere of the brain in the case of M. D., showing the position of the lesion (the dark area to which the letter *T* points.)
The letter *F* points to the frontal and the letter *O* to the occipital end of the brain; *R* to the fissure of Rolando, and *S* to the fissure of Sylvius; *T* to the tumour.

During the course of a bad attack of typhoid, the mouth and tongue become very dry; and the teeth and gums are apt to be covered with sores. This condition of the mouth is often a source of great discomfort to the patient. During the whole course of the disease, and especially during the later stages, when the dryness of the mouth is most marked, the nurse should attend to the condition of the mouth; it may be washed out from time to time with a glycerine and water, claret and water (this I have found is often very grateful to the patient), or with some weak antiseptic solution.

The next point in the treatment of typhoid fever to which I wish to refer, is the administration of antipyretics; but as this question is of very great importance, and as its consideration will require a considerable amount of space, I must defer what I have to say about it until the next issue.

(To be continued.)

IV.—CASE OF LARGE SARCOMATOUS TUMOUR, PRESSING UPON THE MOTOR AREA OF THE BRAIN, AND CAUSING EXTENSIVE DESTRUCTION OF THE NERVE TISSUE, IN WHICH THERE WAS ABSOLUTELY NO PARALYSIS.

M. D., æt. 20, single, shop girl, was admitted to the Newcastle-on-Tyne Infirmary, on 5th February 1877, complaining of headache, vomiting, and giddiness.

Previous History.—When nine years of age she fell and hurt her head; the injury was a severe one, and was followed by vomiting. With this exception, she has never been laid up until the present attack. It commenced three years ago with headache. Eight months ago, the headache got worse, and she vomited occasionally. Two months ago, she had to leave her situation. She states she has never had a fit, but that she has more than once tumbled off her chair when sitting, in consequence, she thinks, of giddiness. She has only menstruated once, and that was two years ago. She knows no

cause for her illness. There is no suspicion of syphilis.

The Family History is good.

Present Condition.—She is a well-developed and well-nourished girl; and, with the exception of a slightly coated tongue, is, as regards the circulatory, respiratory, alimentary, and urinary systems, perfectly healthy.

She seems intelligent, but her friends say that she is very much quieter than she used to be. The face at times has a somewhat dusky, congested hue. The eyes are unusually prominent, but have always been so. The headache is severe, and is worse at night; the pain is referred to the forehead and to the vertex. There is no tender spot on the surface of the cranium.

Special Senses: Sight.—The pupils are equal and moderately dilated. Vision is so perfect that she can read with ease the smallest type, and yet the ophthalmoscope shows marked double optic neuritis. *Hearing.*—The skull sounds are not heard in the right ear. *Smell* is imperfect in both nostrils, but this is probably only temporary, the result of iodism. *Taste* is perfect.

Speech is natural. *There is no trace of paralysis.* The temperature is normal.

Diagnosis.—An intracranial tumour. This opinion was based on the headache, vomiting, and above all the optic neuritis. There were no localising symptoms; the seat of the tumour, therefore, could not be defined.

Treatment.—Full doses of iodide of potassium, and anodynes for the headache.

Progress of the Case.—On 18th and 19th January, she vomited frequently, and the headache was intense. Croton chloral, 10 grains three times daily, was prescribed.

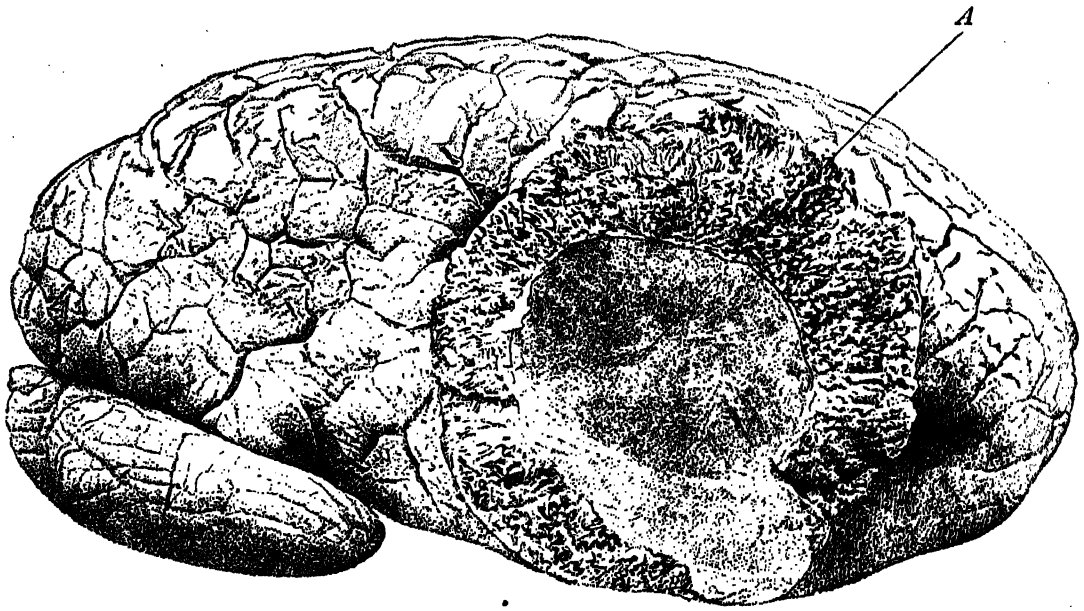


FIG. 94.—The outer surface of the right hemisphere of the brain in the case of M. D. (Large Sarcoma on the surface which had produced extensive atrophy and destruction of the motor area), showing the tumour *in situ*. (Copied from a photograph, and somewhat reduced in size.)
The letter *A* points to the outer surface of the dura mater over the centre of the tumour.

On 28th January, she was very much easier; indeed, she felt so well that she expressed a wish to go home.

On 5th February, there was some return of the headache, but she said it was nothing; and that she felt quite able to go home the next day as she had arranged.

On 6th February.—At 1.30 A.M. my clinical clerk, Mr Tait, was suddenly called to see her. He found that she had just died in a convulsion.

The Autopsy was made eleven hours after death. The head only was examined. The

scalp was natural. The lower part of the right parietal bone, the inferior frontal sub-division of the right frontal bone, and the upper part of the squamous portion of the right temporal bone, were reduced to half their normal thickness. The dura was not adherent to the bone, but was firmly attached to the subjacent membranes in the right inferior frontal and inferior parietal areas. On removing the dura, a large tumour was seen to be situated in these regions. It seemed to spring from the commencement of the right Sylvian fissure, and had destroyed the posterior half of the inferior frontal convolution, the lower half of the ascending parietal convolution, the lower half of the ascending frontal convolution, and the outer half of the island of Reil on the right side. It had also invaded the superficial surface of the posterior half of the middle frontal convolution, the anterior part of the supra-marginal convolution, and the anterior part of the superior sphenoidal convolution. (See Figs. 93, 94, and 95.)

The tumour measured from before backwards 3 inches, from above downwards 3 inches, and from without inwards 1½ inches.

The surface of the tumour, where it was unadherent to the dura, was of a reddish colour, and had a striated, granular appearance. It seemed to be made up of delicate fibre bundles. The margins of the tumour were very thin, and seemed to be gradually applying themselves to and invading adjacent convolutions. The section of the tumour was of a dark purple colour, granular in appearance, and soft and friable. After hardening in spirit it was readily separated from the subjacent brain tissue, and was found to be surrounded by a delicate fibrous capsule. Several large vessels passed into and out of the tumour. On *microscopical examination*, it was found to be a small spindle-celled sarcoma. The tumour was very vascular, but there were no hæmorrhages. The convolutions of the left hemisphere were much flattened, and the sulci effaced, but with this exception, those portions of the brain which were not directly invaded by the tumour, were quite healthy.

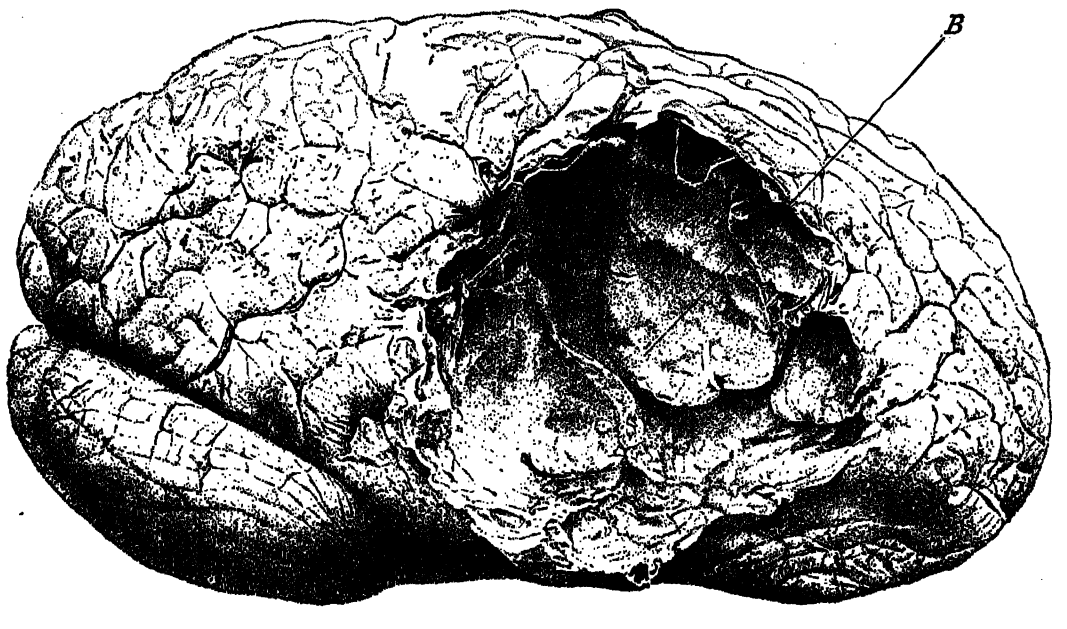


FIG. 95.—The outer surface of the right hemisphere of the brain in the case of M. D. (Large Sarcoma on the surface which had produced extensive atrophy and destruction of the motor area), showing the brain after the tumour was removed. Copied from a photograph, and somewhat reduced in size.)
The letter *B* points to the extensive depression on the surface of the brain, into which the tumour filled.

Studies in Clinical Medicine.

FRIDAY, FEBRUARY 7, 1890.

CONTENTS.

I.—EXOPHTHALMIC GOITRE.

Man; aged 43; labourer; Oct. 9, 1889, and Jan. 11, 1890. *

Dr B. What do you complain of?

Patient. Palpitation and shortness of breath.

Dr B. How long have you been ill?

Patient. About a year.

Dr B. Have you ever had rheumatism?

Patient. No.

Dr B. Is there anything else the matter with you except the beating of the heart and the shortness of breath?

Patient. I feel very weak, and I have no appetite.

Dr B. Are your eyes more prominent than they used to be?

Patient. I have not noticed that.

Dr B. Is your neck swollen?

Patient. I have not noticed.

Dr B. Please let me see your neck.

On examination, a well marked, though not very great, enlargement of the thyroid was found to be present; the eyes were markedly prominent; the pulse numbered 130-140 per minute; the arms and body generally were affected with a fine, rhythmical, vibratory tremor; the patient looked thin, ill, and nervous; and the skin, more especially of the face, was of a dull, earthy, yellow colour.

Dr B. (to the Students.) The case is a very typical and characteristic one. The patient is suffering from Exophthalmic Goitre or Graves' disease—Basedow's disease as it is termed in Germany.

Exophthalmic goitre is a very interesting affection—both clinically, because of the great diversity of the symptoms which it presents; and pathologically, because of the difficulty that there is in explaining the exact manner in which the symptoms are produced. Our knowledge, both of the morbid anatomy and pathology, or the pathological physiology as I am in the habit of terming it, of Graves' disease is still very imperfect.

Exophthalmic goitre is a disease which occurs, as you are aware, chiefly in the female. Males are occasionally, but much less frequently, affected by it. Different observers are not agreed as to the exact relative frequency with which the two sexes are affected; but probably it is not far from the truth to say, that the disease is four or five times more frequent in the female than in the male.

The disease may occur at almost any age; but it is far more frequent during active sexual life than at any other period. The most common period for its development, if I may judge from my own experience, is, in women, between the ages of 15 and 30; and in men, between the ages of 30 and 45. Cases have, however, been observed in children. If I remember rightly, there is one case recorded in

* The account of the case is made up of the remarks made at two Clinics. A good deal of matter has also been added.

which the patient was a child aged two and a half years; and Charcot, in his *Leçons du Mardi*, mentions a case in an old man of 68 years.

The disease usually begins gradually, and lasts for a number of years; it is, in most cases, a chronic affection; cases have, however, been recorded in which the symptoms of Graves' disease have very rapidly manifested themselves after a sudden fright or profound emotional disturbance, and in which, after being present for a short time, they have rapidly disappeared. Cases of this kind are, however, quite exceptional. In the great majority of cases, the development is slow and gradual, and the course chronic.

The exciting cause of the condition seems in many cases to be a fright, profound emotional disturbance, grief, anxiety, or mental strain of some sort or another. Occasionally, the exhaustion produced by acute illness seems to be the exciting cause of the condition. In a few cases, there is a history of head injury. Anything which lowers the nerve tone, and produces exhaustion and debility, may probably, in persons who are predisposed to be affected with exophthalmic goitre, act as an exciting cause of the condition. Many of the patients who are affected with exophthalmic goitre are anæmic; and almost all observers are agreed that anæmia may play some part in the production of the condition.

In some cases, it may, I think, be doubted whether the conditions, which I have just mentioned as the common exciting causes, are the primary cause of the disease. It is probable, I think, that in some cases, at all events, in which a fright or emotional disturbance seems to have been *the* exciting cause of exophthalmic goitre, that the disease was already present, but in such a slight and modified form as to be unnoticed by the patient; and that the fright or emotional disturbance did not actually produce, but only aggravated the symptoms and brought them into such prominence, as to make them distinctly perceptible to the patient. At all events, we must, I think, allow that either the disease, or the tendency to the disease, already existed in a latent form, and that the fright, or other exciting cause, whatever it may happen to have been, upset the balance of those parts of the nervous system in which the lesion (or, perhaps, to speak more cautiously, the functional disturbance) which is the cause of exophthalmic goitre is situated.

The same question arises in connection with the etiology both of chorea and paralysis agitans. In both of these diseases, a fright or other profound mental shock seems, not unfrequently, to be the direct exciting cause of the condition.

There can be no question that many of the subjects of exophthalmic goitre inherit a tendency to nerve disease. In a few cases, exophthalmic goitre appears to have been handed down as a direct inheritance from parent to child; and cases have been reported in which several members of the same family have been affected. But such direct inheritance is extremely rare. In the great majority of cases, in which a patient who is affected with exophthalmic goitre inherits a tendency to nerve disease, the parents have been affected with some other form of nervous malady; the mother, for example, may have been simply what we term nervous, or hysterical; or the father may have been a drunkard; or the parents or some other near relative of the patient may have been insane, or epileptic.

Exophthalmic goitre differs, of course, in this respect in no way from many other—most other—nervous affections. In the great majority of cases in which nervous disease is inherited, the form which the disease takes in the child is different from that which it had in the parent. The mother may be "nervous" or hysterical, or the father may be "peculiar" or a drunkard, while one of the children may be epileptic, another eccentric, a third weak-minded, a fourth actually an idiot, and so on; or the mother may be "nervous" or eccentric, her daughter hysterical, her grandchild insane or epileptic.

It is very rarely indeed the case that the mother or other near relative of a patient affected with exophthalmic goitre has also suffered from Graves' disease, though it fre-

quently happens that the persons affected with exophthalmic goitre inherit a marked neuropathic tendency.

Dr B. (*to the Patient*). Can you blame anything as the cause of your illness?

Patient. No.

Dr B. You were not laid up with any illness before you began to suffer from the palpitation?

Patient. No.

Dr B. You have not had anything to upset you? Any fright, worry, or anxiety?

Patient. No.

Dr B. Do you know if your parents were nervous people?

Patient. No; I do not think so.

Dr B. (*to the Students*). The three great characteristic symptoms of Graves' disease or exophthalmic goitre are increased frequency of the heart's action, enlargement of the thyroid, and prominence of the eyeballs. But these, as we shall presently see, are by no means all of the symptoms which are usually present. In a very considerable number of cases, a characteristic tremor, the special features of which I shall presently describe more in detail, is observed. Indeed, so constant is the tremor, that Charcot includes it, with the abnormal frequency of the heart's action, the enlargement of the thyroid, and the prominence of the eyeballs, as one of the primary or fundamental symptoms of the disease.

I append a Table, which is copied from Charcot, in which the many different symptoms, which may be present in cases of exophthalmic goitre, are enumerated and classified. I will afterwards refer in detail to some of the more important of these symptoms:—

Table showing the series of symptoms which may be present in cases of exophthalmic goitre or Graves' disease—(after Charcot).

PRIMARY or CARDINAL.		Increased frequency of heart's action (asystole). Goitre. Exophthalmos. Rythmical vibratory tremor.
SECONDARY.	*Digestive organs.*	Vomiting. Bulimia; Sudden fits of hunger. Jaundice.
	Respiratory organs.	Cough. Increased frequency of respiration.
	Nervous system.	Angina pectoris; Neuralgia. Paralysis; Von Graefe's symptom; Peculiar form of paraplegia Difficulty of convergence (Möbius). Convulsions; Epileptiform crises. Psychical modifications (emotional, &c.).
	Integumentary system.	Vitiligo; Urticaria; Pigmentary patches. Sweatings; Sensations of heat. Diminished electrical resistance.
	Urinary system.	Polyuria; Albuminuria. Glycosuria.
	Generative system.	Menstrual derangements. Impotence.
	General.	Anæmia more or less profound—Cachexia—Œdema of the lower extremities due to asystole.

The great primary (fundamental) symptoms of exophthalmic goitre are, then, increased frequency of the heart's action, enlargement of the thyroid, and prominence of the eyeballs; and to these Professor Charcot would add the characteristic tremor, to which I will presently refer.

The appearance which a patient, who is affected with exophthalmic goitre, in its typical, classical, and fully developed form, presents, is highly characteristic. Graves' disease is, in fact, one of the few diseases which (but only when it is typical and fully developed) one can recognise at a glance. The physiognomy of the patient is quite characteristic or pathognomonic of the condition.

The remarkable prominence of the eyeballs, and the startled, staring, and in some cases almost savage appearance of the face, are very peculiar.

These features, and the obvious enlargement of the thyroid, are admirably shown in the highly artistic, and, at the same time, absolutely truthful portrait of a patient who presented herself at the Clinic some three years ago. (See fig. 96, and note that the plate is erroneously numbered 93 instead of 96.)

It must not, however, be supposed that one can recognise *all* cases of exophthalmic goitre at a glance. It is by no means very uncommon to meet with cases in which there is no exophthalmos. Cases are also sometimes met with in which there is no enlargement of the thyroid. The symptom which is constant, is increased frequency of the heart's action. I shall presently describe in detail the exact characters of the cardiac derangement, and of the other fundamental symptoms of the disease. But before doing so, let me again emphasise the fact, that cases are by no means very uncommon in which the disease is developed, as it were, in a fragmentary or imperfect manner. I have myself met with a considerable number of these rudimentary a-typical forms of exophthalmic goitre (*fruste*, as the French writers term them). If I may judge from my own experience, these rudimentary cases of Graves' disease are more common in men than in women. Men, as every one knows, are much less frequently affected with exophthalmic goitre than women; and it would appear (at all events, so far as I am able to judge from my own experience) that when men do become affected with Graves' disease, that the clinical picture is apt to be imperfect; in men, some of the symptoms, some of the primary symptoms which are usually seen in women—I allude more especially to the prominence of the eyeballs and the enlargement of the thyroid—are apt to be wanting. It must not, however, be supposed that in men the disease is less severe, or less serious, than in women. In fact, the contrary is, in my experience, the case. In the male, the disease is, I think, as a rule—I speak of course generally—more severe than in the female. I can recall several well marked a-typical or rudimentary cases of exophthalmic goitre in males, which have come under my own observation. The first case of the kind which I clearly recognised—only, however, after its true nature had been pointed out by Sir William Jenner—was that of a gentleman whom I saw in consultation with my friend Dr Croom, some ten years ago. He was a dark skinned, very nervous man, of 45 or 50 years of age. His pulse was habitually very frequent, and he was not only nervous, but affected with a very distinct tremor, which I now know to be very suggestive of the disease. Neither Dr Croom nor I could make up our minds as to the exact nature of the condition. Some little time after I saw the patient, he consulted Sir William Jenner, who diagnosed Graves' disease. During the past ten years, I have seen several cases of the same kind—cases of rudimentary Graves' disease in the male,—in which there was neither enlargement of the thyroid nor prominence of the eye-balls. One of these cases—the patient was a clergyman, under the care of Dr Millard—had been seen by Professor Charcot, who had given a written diagnosis of Graves' disease. In France, it is, I believe, the custom after a consultation to draw up a written statement of the facts of the case, with the diagnosis, prognosis, and treatment. This statement,

FIG. 93.—Exophthalmic goître.

is signed by the medical men who see the patient. Such a method of procedure is not followed in this country; but I can conceive that its adoption would be eminently conducive to accuracy, and would prevent a great deal of loose statement. Another case of rudimentary Graves' disease is at present under my observation. He occasionally comes to the Clinic as an out-patient; and I will take the opportunity of bringing him at some future time under your observation.

Let me repeat that in some cases of Graves' disease, the primary symptoms, on account of which the name exophthalmic goitre has been given to it, may be wanting. In some cases, there is no exophthalmos; in others, there is no enlargement of the thyroid; in others again, there is neither prominence of the eyeballs nor enlargement of the thyroid. Increased frequency of the heart's action is, however, always present. It is *the* symptom which is never (permanently) absent. Without increased frequency of the heart's action, a diagnosis of Graves' disease is (so far as we at present know) never justifiable. You must have the increased action of the heart, or you are unable to diagnose Graves' disease.

Let me now consider the individual symptoms in more detail.

I will first refer to *the disturbance of the heart and circulation*, since they are the most constant and, in most cases, the first symptoms which are developed. I have already stated that a diagnosis of Graves' disease is never warranted unless increased frequency of the heart's action and of the pulse is present.

In the great majority of cases of Graves' disease, the frequency of the heart's action is greatly above the normal. The pulse usually numbers 120, 130, or 150 in the minute. In some cases it is indeed much more frequent. I have in several instances noted a pulse of 180 in the minute. Exceptionally the heart's beats are not much accelerated. In some cases, the pulse only numbers 100, or even 90 in the minute. I refer, of course, to the frequency of the pulse where the disease is fully developed. At the beginning of the disease, and still more at the end, when the symptoms are subsiding, as they not unfrequently do in the course of time and as the results of treatment, the pulse may, of course, number only 90 or even less in the minute. It is very important to note, that diminished frequency of the pulse is the most certain sign of improvement which we possess. *Vice versâ* a very quick pulse, and an increasingly quick pulse, is a bad sign. The behaviour of the pulse under treatment is our most important guide for prognosis.

In cases of exophthalmic goitre, the increased frequency of the heart's action is at times much more noticeable than at others. Naturally, as one might expect, the pulse frequency is much increased by any emotional disturbance or excitement. As in all nervous persons, the pulse frequency is often much increased when the patient comes before the doctor. This point must, of course, be allowed for. The subjects of Graves' disease are eminently nervous. The most striking fact in the disease is perhaps the extreme irritability of the heart. The heart seems to run loose; it would appear that its action is no longer restrained or reined in by the controlling and inhibitory influence of the vagus; the accelerating influence of the sympathetic has the upper hand; and conditions (irritations and stimuli, emotional or other), which produced little or no effect upon a normal heart, appear to lash it into an altogether unnatural and furious activity.

It is important, however, to note that in some cases of Graves' disease, the increased frequency of the cardiac action is paroxysmal and intermittent. I have never seen a case of this kind myself, but Charcot states that such cases do occur. Cases of this kind, more especially if there was neither enlargement of the thyroid nor prominence of the eyeballs, might naturally give rise to great difficulty in diagnosis. If the physician should happen to see the case during an a-frequent interval (*i.e.*, at a time when the paroxysm of accelerated action, so to speak, is not present), a positive diagnosis could not (in the absence of exophthalmos and thyroid enlargement) be made. Such a difficulty in diagnosis is not, however, likely to occur. It is, I fancy, more

theoretical than practical. In the vast majority of cases of exophthalmic goitre, the agitation and nervous excitement, which the patient feels under the examination of the physician, would, even if the pulse were at times slow, set the heart off at a gallop, and excite a paroxysm of increased frequency.

Though the subjects of exophthalmic goitre usually complain of the increased frequency and undue irritability of the heart's action, and often, too, of a feeling of throbbing or pulsation in the vessels of the neck and of the head, and in some cases of the body generally, they do not, as a rule, appear to suffer in the same degree, and to feel the same sensation of anxiety and dread, which patients who are *suddenly* attacked with a paroxysm of ordinary functional palpitation do.

I have underlined the word suddenly, for the sudden, intermittent, unaccustomed, and paroxysmal character of an ordinary attack of palpitation seems to me to be a sufficient explanation of the very different character of the subjective sensations which the patient experiences in the two cases. I should fancy, if the heart of a healthy individual were suddenly made to beat in the way in which the heart of a patient with Graves' disease is in the habit of beating, that the healthy individual would experience all the alarming symptoms which are usually associated with an ordinary attack of functional palpitation. In exophthalmic goitre, the derangement of the heart is, as a rule, gradually and slowly developed; the increased frequency and the undue irritability of the heart's action continue for months or years; they become, as it were, part and parcel of the individual. It is natural, therefore, to suppose that when a heart, whose action is habitually increased and unduly irritable, becomes temporarily still more accelerated —accelerated to a degree which, in a normal individual, would be attended with all the subjective sensations of a violent attack of palpitation—that the subjective sensations, which attend an attack of ordinary palpitation, would be experienced in a very modified degree.

The influence of habit is a very important influence in disease as well as in health. It is truly astonishing to note how nature, or rather the sensorium, after a time may come to ignore—to adapt itself, as it were, to new and even most remarkable pathological conditions. Innumerable illustrations in support of this proposition might easily be given. I shall only mention one. Most medical men, knowing the risk of sudden death which aortic regurgitation may entail, would, it may safely be affirmed, be very much disturbed if they were told all at once that they were the subjects of that disease. In a small proportion of cases, the dread of sudden death would perhaps continue as an ever-present and ever-distressing factor in their future lives; but it may safely be affirmed that in the large majority of cases, although the cause (the aortic regurgitation) would continue, and the knowledge that the cause was incurable would continue, yet the dread of sudden death would, after a little time, be no longer entertained, though the probability of the occurrence of sudden death might still be clearly appreciated.

In exophthalmic goitre the heart's action and the pulse are, as a rule, regular. In the later stages of the disease, and as the result of cardiac failure and dilatation, the heart's action may become irregular or intermittent. Irregularity of the pulse is sometimes present, though rarely, as the result of organic valvular disease.

The chief characters of the pulse, the increased frequency, the regularity, the small volume, and tendency to dicrotism, which are so frequently seen in cases of exophthalmic goitre, are well shown in fig. 97, which was taken from the patient who is represented in fig. 96 (erroneously numbered 93).

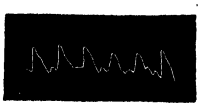


FIG. 97.—Pulse tracing from the case of exophthalmic goitre represented in Fig. 96.

It is a very important fact, that in the great majority of cases of exophthalmic goitre the heart is unaffected with organic disease

In the later stages of some cases, a certain amount of dilatation, or dilatation and hypertrophy, may be present; but this is by no means always so. It not unfrequently happens that after death the heart is found to be little if at all larger than normal.

More than one case has come under my own observation, in which, during the later stages of the case, the heart was considerably enlarged, and very definite evidence both of mitral and tricuspid regurgitation was present. It is by no means very uncommon in the later stages to find œdema of the feet developed.

In almost every well-marked case of exophthalmic goitre, some shortness of breath on exertion, which is probably of cardiac origin, is complained of.

When we examine the heart of a patient who is suffering from well marked exophthalmic goitre, we usually find that the area of visible impulse is increased; and in many cases, an abnormal degree of epigastric pulsation is present.

The area of deep cardiac dulness is not, as a rule, much, if at all, enlarged.

The action of the heart is sharp and flapping in character; the first sound is usually short in duration and accentuated in tone. It is said that the cardiac sounds may in some cases be so much accentuated as to be auto-audible (*i.e.*, heard at some distance from the patient, and without the aid of the stethoscope). A systolic murmur can very frequently be heard at the base, and in some cases in the mitral area. In many cases, these murmurs are doubtless hæmic; in some, they are perhaps due to the altered character of the cardiac contractions, and are independent of anæmia; in a few cases, systolic mitral or tricuspid murmurs, the result of cardiac (ventricular) dilatation, are present; mitral and tricuspid incompetence are, however, comparatively rare, even in advanced cases. In exceptional cases, exophthalmic goitre is complicated with organic valvular lesions, the result of endocarditis. The subjects of Graves' disease seem very rarely to be attacked with acute articular rheumatism. Only one well-marked case of this kind has come under my own observation. In the case to which I refer, a girl, some sixteen or seventeen years of age, who presented all the typical symptoms of exophthalmic goitre, had a very severe attack of acute rheumatism, in which both endocarditis and pericarditis developed.

Exaggerated pulsation in the vessels of the neck is a striking symptom in many cases of exophthalmic goitre; the pulsation may be "hammering" in character. Very distinct pulsation and thrill can usually be felt over the enlarged thyroid. The veins of the neck may be unduly prominent; and in the advanced stages of some cases, pulsation in the veins of the neck can be observed. In one case of the kind, I satisfied myself that the venous pulsation was due to tricuspid incompetence and not merely the result of communicated pulsation from the carotid artery.

Blowing systolic murmurs can be heard in the carotids, over the enlarged thyroid, and over the prominent eyeballs. In many cases, and this is only what we would of course expect from the frequency with which anæmia is present, a well-marked venous hum is audible at the root of the neck.

I have already mentioned the sensation of pulsation which the patient often feels in the vessels of the neck and head, and it may be of other parts of the body. Tinnitus, headache, and in less frequent cases vertigo may be complained of.

The exact cause of the increased frequency of the heart's action is a matter of dispute. According to one view, it is the result of over-action (irritation) of the sympathetic; and according to another, of under-action or diminished action (paralysis) of the vagus. The latter view seems to me the more probable. I shall return to the subject in discussing the pathology of the disease.

The enlargement of the thyroid, which, in typical and fully developed cases of the disease, is a striking feature, may develop before, or after, or simultaneously with the exophthalmos. It is usually (probably always) developed later than the increased frequency of the heart, though it may be the first thing which attracts the attention of the patient.

In some cases, as I have already mentioned, the thyroid does not enlarge; and in such cases exophthalmos is usually also wanting.

The enlargement of the thyroid is only moderate in degree. The gland does not attain a large size, as in ordinary goitre. The enlargement, in the earlier stages at all events, is a soft enlargement, and is usually thought to be due to dilatation of the thyroidal vessels. I see, however, that this statement has recently been called in question. At a recent meeting of the Pathological Society of London (reported in the *British Medical Journal* of January 25), Dr James Berry showed a specimen of enlarged thyroid from a case of exophthalmic goitre, and directed attention to the fact that the thyroidal arteries were of small size; he further stated, that this and other facts show that the gland in this disease is not nearly so vascular as is often supposed.

I have already directed attention to the fact that pulsation and thrills can be felt, and blowing murmurs heard over the enlarged and soft thyroid gland. Enlarged and dilated veins may sometimes be seen over the enlarged thyroid. (See fig. 96, erroneously numbered fig. 93.)

In the later stages of the disease, the enlarged thyroid not unfrequently becomes harder; this is doubtless due to hyperplastic changes in the gland tissue.

All parts of the gland are as a rule enlarged; but the enlargement may be more on one side than on the other. As a rule, the right lobe is more enlarged than the left. It has been suggested, that this is due to the fact, that the right vagus, which seems to have a more powerful inhibitory influence on the heart than the left vagus, is more affected in exophthalmic goitre than its fellow on the opposite side.

A remarkable case has been reported and figured by Dr Burney Yeo, in which the exophthalmos was limited to the right eye, and in which the right lobe of the thyroid was alone enlarged.

The enlargement of the thyroid, like the prominence of the eyeballs, may undoubtedly vary from time to time; it may, I believe, in some cases be seen to increase slightly during emotional excitement. This and the soft character of the enlargement, together with the fact that pulsation and thrills can be felt over it, seem to me to be strong arguments in favour of the increased vascularity of the organ.

The next symptom to which I wish particularly to refer is the *prominence of the eyeballs.* As I have already remarked, the exophthalmos is usually developed simultaneously with the enlargement of the thyroid, and later than the increased frequency of the heart. The degree of prominence varies considerably in different cases. In exceptional cases, there may be no exophthalmos. In the majority of typical cases, the prominence of the eyeballs is very noticeable. When the exophthalmos is great, and especially when the upper lid is at the same time spasmodically retracted, the countenance acquires a wild, staring look, which is well shown in fig. 96 (erroneously numbered 93 in the plate). The protrusion of the eyeballs may be so great as to expose the insertions of the recti muscles; it has even been stated that the exophthalmos may be so extreme that the eye becomes dislocated out of the orbit, and has to be pushed back into its place by the fingers. In rare cases, the exophthalmos is unilateral. Dr Burney Yeo has recorded and figured a remarkable instance of this kind. The degree of prominence is undoubtedly apt to vary from time to time. Under emotional excitement, the eyeballs may become more prominent. In some cases, in which the exophthalmos appears to become greater under excitement, there is in reality little difference in the degree of actual protrusion; but the increase of the exophthalmos is apparent only, and due to the fact that under emotional excitement the upper lid becomes more retracted, and a larger portion of the sclerotic is exposed than under ordinary conditions.

It has been stated that the eyeball itself is actually enlarged in some cases of Graves' disease; but if such is the fact, the degree of enlargement is certainly inconsiderable. In some cases in which the exophthalmos is great the lids are unable, when closed, to completely

cover the ball. Under such circumstances the eye may become inflamed, and conjunctivitis or ulceration of the cornea be produced. Dr George Berry states that a certain degree of anæsthesia of the cornea is found in some cases; and he suggests that in cases of this kind there is probably a greater tendency to ulceration of the cornea than in others. Anæsthesia of the cornea is consequently, he thinks, a symptom which deserves special attention.

In the great majority of cases of exophthalmic goitre (provided that the cornea is not ulcerated—and this condition is, as I have already stated, extremely rare) vision is unaffected. Pulsation in the retinal arteries can be seen with the ophthalmoscope in some cases, but the most constant alteration in the fundus seems to be dilatation of the retinal veins.

The pupils are, as a rule, normal; it is certainly exceptional to meet with any marked degree of dilatation. This is a fact of great importance with regard to the pathology of the disease; for if, as some authorities have supposed, the condition is due to irritation of the cervical sympathetic, dilatation of the pupil ought to be present. In some cases, the pupils are unequal as regards size; but, as a rule, no marked difference is observed.

Increase of the fat at the back of the orbit, and dilatation and engorgement of the vessels at the back of the orbit, are usually considered to be the chief causes of the exophthalmos. Spasm of the fibres of Müller's muscle, is another condition to which the prominence of the eyeballs has been attributed.

Before leaving the condition of the eyeballs, I must not forget to refer to certain other ocular symptoms which may be present. In many cases of Graves' disease, the upper eyelid is spasmodically contracted, and the aperture between the eyelids is wider than normal. The spasmodic retraction of the upper lid, is apt in my experience to be increased under excitement; as well seen in fig. 96 (93). It is the combination of the exophthalmos with the retraction of the lids, which gives the countenance the staring, frightened appearance which is so characteristic of the disease. The retraction of the upper lid, which seems to be due to spasm of Müller's muscle, goes by the name of Stellway's sign. Spasmodic retraction of the lids, is supposed to be the cause of the absence of reflex blinking, which is observed in some cases of Graves' disease. Possibly in some cases, in which the normal reflex blinking movements are more or less completely absent, anæsthesia of the cornea and conjunctiva is the cause of the condition (*i.e.*, of the absence of blinking). Dr George Berry states, that "this retraction of the upper lid is not met with in other forms of exophthalmos, and is, therefore, a point of diagnostic importance, in the cases where the exophthalmos is confined to one side, or not accompanied in a marked degree by the other symptoms of Graves' disease. It frequently," he says, "exists at a time when there is little or no protrusion of the eyes, and calls attention to the condition of the circulation and thyroid."*

I remember that I was very forcibly impressed with a marked spasmodic and intermittent contraction of the upper eyelids in two cases which came under my observation a number of years ago. One was an out-patient at the Newcastle Infirmary, the other came under my notice at the Cowgate Dispensary in this city. Both patients were males, and both were suffering from symptoms indicative of mental derangement. In neither case was the thyroid enlarged, nor the eyeballs prominent. Possibly these patients were suffering from Graves' disease, in its rudimentary or imperfectly developed forms. It is well known—Dr Savage amongst other writers having published cases of this kind—that insanity and exophthalmic goitre are sometimes associated.

Another symptom which is present in some, though by no means in all, cases of exophthalmic goitre, was first described by the celebrated German oculist Von Graefe; it is known as Von Graefe's sign. It consists apparently in a want of co-ordinate action between the eyeball and the upper lid. If you stand in front of a healthy individual, and make him "fix" and follow your finger with his eye, as you move it slowly from above the horizontal line of his vision downwards

* *Diseases of the Eye*, page 385.

towards the ground, you will see that, as his eye moves downwards in following the movement of your finger, the upper lid moves simultaneously downwards with the ball. In health, the downward movement of the eyeball is accompanied by a simultaneous and exactly co-ordinated movement of the upper lid. Now, in some cases of Graves' disease, the upper lid fails to follow the eyeball downwards in this steady co-ordinate manner. The movement of the lid, as it were, hangs fire altogether, or follows the downward movement of the eyeball in an uncertain and jerky manner. This symptom is very characteristic of exophthalmic goitre, though not absolutely pathognomonic of that condition. We recently had at the Clinic a patient who presented none of the other symptoms of exophthalmic goitre, in whom Von Graefe's sign was present in a very marked degree. I will publish the brief notes of this case in the next number of these *Studies*.

FIG. 98.—Pulse tracing from the case of exophthalmic goitre described in the text, shewing the presence of the characteristic tremor.

FIG. 99.—Tracing of the tremor, taken at the wrist, from a case of exophthalmic goitre.

FIG. 100.—Sphygmogram from a case of exophthalmic goitre, shewing the presence of the characteristic tremor. The regularity of the pulse curves is broken by the tremor.

I repeat, that Von Graefe's symptom, viz., the non-descent of the upper lid (or, to speak more accurately, the non-simultaneous or incoordinate descent of the upper lid), when the eyeball is made to travel from above downwards, is a highly characteristic sign of exophthalmic goitre. It is, however, by no means constantly present.

Von Graefe's sign was well developed in the case which forms the text of these remarks, and was demonstrated to the students who were present at the Clinic.

In some cases of exophthalmic goitre, inability to converge for near objects has been noted. Ophthalmoplegia externa, or paralysis of all the external muscles of the eyeball, has also been observed. A remarkable case of this kind has been described by Dr Bristowe. A certain degree of weakness or paralysis of the recti muscles may, perhaps, be due to overstretching or fatty changes (which are said not unfrequently to be present in exophthalmic goitre); but the presence of such a marked degree of paralysis as was present in Dr Bristowe's case, is highly suggestive of a central lesion, and affords corroborative evidence that the lesion in cases of exophthalmic goitre is situated in the nerve centres (medulla oblongata) rather than in the gangliated cord of the cervical sympathetic.

A further symptom, which Charcot includes under the primary or fundamental characteristics of Graves' disease, is *muscular tremor*. This symptom has been more particularly studied by M. Marie, to whom and to the illustrious Charcot the profession is indebted for much of our recent knowledge of the symptomatology of exophthalmic goitre. The characters of the tremor are peculiar. The tremor is due to fine, rhythmical, muscular movements which occur with great rapidity (8 or 9 per second). According to M. Marie and Professor Charcot, the rapidity of the muscular movements which produce the tremor of Graves' disease, is nearly twice as great as in paralysis agitans (in which the muscular contractions number 4 or 5 per second). The tremor may, and usually does, affect the muscles of the lower limbs and of the trunk, as well as the muscles of the upper extremities. In many cases, the whole body can be felt to shake if the hand is placed upon the top of the head or upon the shoulder. Charcot states that this peculiar muscular tremor may be one of the first

symptoms to be developed; and that it is of great diagnostic importance. In some of the imperfectly developed or rudimentary forms of the disease, in which there is no enlargement of the thyroid and no exophthalmos, but in which there is a notable and permanent increase in the frequency of the pulse, the presence of this characteristic, fine, quick, rhythmical tremor confirms and greatly strengthens the diagnosis. Charcot directs special attention to the fact that in Graves' disease the individual digits do not tremble. In this respect, the tremor of exophthalmic goitre differs from alcoholic tremor and from the tremor of progressive general paralysis; in both of these affections, the tremor, as in Graves' disease, is a very rapid tremor (8 or 9 per second).

Since I became acquainted with the observations of M. Marie and Professor Charcot, I have directed especial attention to the presence of this characteristic tremor in cases of exophthalmic goitre. My own observations are not sufficiently numerous to allow me to generalise upon the matter; but so far as they go, they are entirely in accord with Professor Charcot's views. In three out of four cases of exophthalmic goitre, which I have had the opportunity of observing, in hospital or private practice, during the past year, the characteristic tremor was present. In the case which forms the text of these remarks, the peculiar tremor was very noticeable. Its characteristic features could be well studied by means of the sphygmograph. I append two tracings from this case. Fig. 98 is a pulse tracing, in which the regularity of the pulse wave is seen to be broken by the tremor. Fig. 99 is a tracing taken from the fore-arm, the bottom of the sphygmograph being placed over the centre of the wrist; in it the exact characters of the rhythmical tremor (irrespective of the pulse waves which are shown on the previous tracing, Fig. 98) are well shown. I also add a pulse tracing (Fig. 100) taken from another case of Graves' disease (in which there was neither enlargement of the thyroid nor prominence of eyeballs) in which a combined tracing of the pulse curve and the tremor is shown.

In the next number of these *Studies*, I will direct attention to some of the other symptoms which may be present in exophthalmic goitre; and will consider the diagnosis, prognosis, and treatment.

II.—Case Illustrative of the Differential Diagnosis of Hysteria and Cerebral Tumour; Suspected Vicarious Menstruation.

Girl; age 25; unmarried.

Dr B. (to the Students). Dr Whiting tells me, Gentlemen, that this patient is suffering from symptoms which suggest the possibility of an intracranial tumour. Before coming into the Clinic, I have examined the optic discs. The fundus is perfectly normal. I do not know the nature of her symptoms, for I have not as yet asked her any questions. I see, however, she brings a note from her doctor.

(Dr Bramwell here read the note.)

The doctor's letter gives a very clear account of the case. (Certain parts of the letter, which is quoted in full below, were then read to the students in the presence of the patient and her mother, who accompanied her.)

The letter is as follows:—"A. B.; age 25, complains of headache, and cramps in the hands and feet. She has been affected with headache for nearly a year; at first, the pain was chiefly felt over the left eye; for two months, it has affected the back of the head on the right side. The headache is always worse at night, and is accompanied with cramps in the hands and feet—the right hand is most affected. In the attacks, the hands are clenched and pronate. In the feet, the flexor muscles are chiefly affected. During the attacks of pain, she has an inclination to vomit, but nothing comes up. I have never seen her at night, when the attacks come on, as she lives some distance from me in the country. The house she lives in is old and very damp. Ten

or eleven years ago, when she was about fourteen years of age, I had considerable trouble with her. She became melancholy, after the death of two sisters or brothers from scarlet fever. At that time she had great difficulty in swallowing, because of a spasm in the throat. We had great difficulty in feeding her. In fact, she almost developed into a fasting girl. Her mother says that the spasm in the throat still affects her when the pain is bad. She had commenced to menstruate before her illness of eleven years ago began. The menstruation ceased during the illness. Her mother states that when she was getting better she used to vomit blood monthly (? vicarious menstruation). I am no great hand at the ophthalmoscope, but I don't think there is any optic neuritis. Her urine has a sp. gr. of 1030; it contains urates. She had been treated chiefly with tonics, iron, quinine, strychnine, etc. She follows the occupation of a dressmaker, but has not been able to do much for some time. Her mother and brother, who accompany her, will be able to give you more detailed information. I hope you will find the case of some interest for your Clinic."

Dr B. (*to the Patient*). Is the headache very severe?

Patient's Mother. Yes, it is very bad.

Dr B. She has never vomited?

Patient's Mother. No, but she feels sick.

Dr B. Does the pain come on at any particular time?

Patient's Mother. Yes, in the evening. It usually comes on about six o'clock.

Dr B. How long does it last?

Patient's Mother. Till four in the morning. It is very bad from six to eight o'clock. After that it is not so severe.

Dr B. You say she has a choky feeling in the throat during the attacks of pain?

Patient's Mother. Yes.

Dr B. What are the spasms like?

Patient's Mother (*clenching the fists, and extending and pronating the forearms*). The hands are stretched out like this.

Dr B. The right hand is more affected than the left?

Patient's Mother. Yes.

Dr B. Are both feet affected?

Patient's Mother. Yes.

Dr B. Does she stiffen and arch her back in the attacks?

Patient's Mother. Yes, the back is quite stiff.

Dr B. Does she bend or arch her back, backwards?

Patient's Mother. She makes her back stiff.

Dr B. Does she roll her head from side to side, and throw her arms about in the attack?

Patient's Mother. Yes.

Dr B. Does she ever burst out crying or laughing during the attacks?

Patient's Mother. Not much.

Dr B. Did she ever do so during her former illness?

Patient's Mother. Yes.

Dr B. Has she ever had a fit?

Patient's Mother. No.

Dr B. Does she ever make a large quantity of water after the attacks?

Patient's Mother. I have not observed that.

Dr B. Is she ever unconscious during the attacks?

Patient's Mother. No.

Dr B. Does she complain of anything else?

Patient's Mother. No.

Dr B. She is a very nervous girl?

Patient's Mother. Yes.

Dr B. Before these headaches and spasms commenced, had she anything to upset her or disturb her?

Patient's Mother. No.

Dr B. Is her appetite good?

Patient's Mother. Yes.

Dr B. And her digestion?

Patient's Mother. Yes.

Dr B. Are her bowels quite right?

Patient's Mother. Yes.

Dr B. Is she now quite regular?

Patient's Mother. Yes.

Dr B. You say that she used to vomit blood, when she was ill some years ago?

Patient's Mother. Yes.

Dr B. And you think that vomiting of blood took the place of the monthly flow?

Patient's Mother. Yes, I thought so.

Dr B. How many times did she vomit blood?

Patient's Mother. Three or four times—she coughed it up.

Dr B. What was the greatest quantity of blood that she brought up at any one time?

Patient's Mother. About a table spoonful and a half.

Dr B. Was it light or dark?

Patient's Mother. Dark.

Dr B. Were there any clots in it?

Patient's Mother. Yes.

Dr B. What was the size of the clots? What was the size of the biggest clot?

Patient's Mother. The size of a marble.

Dr B. How long did she continue to cough up blood at a time?

Patient's Mother. Two days.

Dr B. And that occurred three or four times?

Patient's Mother. Yes.

Dr B. Regularly every month?

Patient's Mother. Yes.

Dr B. You are quite satisfied that the blood occurred regularly every month, when she ought to have been menstruating?

Patient's Mother. I thought so.

Dr B. Was she troubled with any stomach symptoms or cough when she was bringing up the blood? Had she any indigestion or pain in the stomach after eating?

Patient's Mother. No.

Dr B. Did you see the blood come up?

Patient's Mother. Yes.

Dr B. What time of the day used it to come up?

Patient's Mother. Usually about twelve or one o'clock.

Dr B. When she was in the room beside you?

Patient's Mother. Yes, she used to be sitting beside me.

Dr B. Did she ever bring up any blood after she became regular again?

Patient's Mother. No.

Dr B. When she became regular, the vomiting of blood ceased?

Patient's Mother. Yes.

Dr B. Did she make a complaint before the blood came up?

Patient's Mother. No.

Dr B. (to the Students). There is a popular notion both amongst the laity and the profession that vicarious menstruation does occur. I must confess that I share that impression. I have certainly from time to time met with cases which have seemed to me to be cases of vicarious menstruation. In this case, the facts which we have just elicited seem to lend support to that view. From what the patient's mother has just told us, I should be inclined to think that this is a case of vicarious menstruation. I see nothing impossible in the occurrence of vicarious menstruation. It is certainly the fact that hæmorrhage may occur into the fundus oculi in cases of amenorrhœa; perhaps, however, it is due to the anæmia with which amenorrhœa is often associated; and if hæmorrhage may occur into the retina, I see no reason why hæmoptysis and other forms of external hæmorrhage may not occur, and take the place as it were of the normal menstrual flow. Some authorities doubt the occurrence of vicarious menstruation. Dr Wilks disputes, if I remember rightly, the occurrence of vicarious menstruation. He has, I think, stated that there is no case on record, which clearly and unmistakeably proves the occurrence of vicarious menstruation.

Cases of supposed menstruation very frequently occur in hysterical cases; and hysterical patients are often, as we know, very deceitful. It is necessary to guard therefore against the possibility of deception, and to be quite sure that the blood is actually coughed up, or vomited, or discharged in a natural manner. In this case, there seems to be no doubt, from the statement of the mother which you have just heard, that the blood was brought up in a natural (not artificial) manner. There is no reason to suppose that it was artificially evacuated.

To clearly establish the occurrence of vicarious menstruation, it is necessary that the case should fulfil certain rigid conditions. In the *first* place, the patient should have been previously regular. In the *second* place, with the arrest of the normal menstrual flow, blood should be actually, and so to speak naturally (not

artificially), discharged from some other surface than the interior of the uterus, regularly every month, and should take the place of the menstrual flow. The vicarious discharge should occur regularly every month instead of menstruation, and should not occur at any other time except when the menstruation ought to have been present. In the *third* place, when the normal menstrual flow is again established, the abnormal or vicarious discharge of blood should cease, and should not again return unless the menstruation is again arrested. And in the *fourth* place, the patient should not, previous to the arrest of menstruation, have ever suffered from a discharge of blood such as the vicarious menstruation consists of. If, for instance, the vicarious discharge of blood is from the lungs, the patient should never have previously been affected with hæmoptysis.

In order to clearly and definitely establish the occurrence of vicarious menstruation, the case must fulfil these conditions. These requirements are rigid. I do not say that no case is a case of vicarious menstruation which does not fulfil these conditions. That is a very different proposition. If, as I believe, vicarious menstruation does occur, it is only reasonable to suppose that many cases—the great majority of cases—will not fulfil the rigid conditions which are required for actual and scientific proof of the condition. But, if there is such a thing as vicarious menstruation, some cases ought to be met with which do fulfil these conditions.

Dr B. (*to the Patient's Mother*). You say that your daughter began to menstruate, and for a time menstruated regularly before her illness eleven years ago?

Patient's Mother. Yes.

Dr B. And that when she became ill she ceased to menstruate?

Patient's Mother. Yes, for two years.

Dr B. During the time that she was ill, she spat or vomited blood on three or four occasions, and at monthly intervals; and you are under the impression that these vomitings of blood took the place of the menstrual discharge?

Patient's Mother. Yes; it was when she was getting better from the illness that the vomiting of blood took place.

Dr B. After she again became regular, the vomiting or spitting of blood ceased, and she has never since vomited nor spat blood?

Patient's Mother. No.

Dr B. Did she ever vomit or spit blood before the three or four occasions on which the discharges of blood from the mouth, which you have told us of, occurred?

Patient's Mother. While she was ill there was a discharge of blood for about a year from the mouth. I thought it came from the gums.

Dr B. (*to the Students*). That statement puts the case on an altogether different basis. It throws grave doubt upon the vicarious menstruation theory. The case obviously does not fulfil the rigid requirements which I have laid down as necessary for proving to demonstration that it is a case of vicarious menstruation. You have just heard that, for about a year before the supposed vicarious menstruation occurred, there was a discharge of blood from the mouth, perhaps from the gums.

Dr B. (*to the Patient's Mother*). Did the discharge from the gums go on continuously, or did it, like the three or four vomitings or spittings of blood, which you have told us of, occur at monthly intervals?

Patient's Mother. It did not occur at monthly intervals; it went on every now and again for a year.

Dr B. More or less continuously for a year?

Patient's Mother. Yes.

Dr B. (*to the Students*). I am afraid, after that statement, that we can hardly consider the case as an undoubted case of vicarious menstruation. It may have been so. But there is no scientific proof that it was so. The evidence is not sufficient to warrant the conclusion that it is a case of vicarious menstruation.

Dr B. (*to the Patient's Mother*). Please take your daughter into the next room. She will be further examined, and you will be then told what to do.

Dr B. (*to the Students*). This girl has suffered from headache for several months; the attacks of headache seem to come on almost every day

at the same time; when the headache is very severe, she has spasmodic attacks; the character of the spasms seems clearly to be hysterical rather than epileptiform; the spasms do not resemble the localised spasmodic movements of Jacksonian epilepsy. In some cases of tumour of the cerebellum, tonic spasms are met with; but, so far as I see, there are no facts in this case to warrant a diagnosis of tumour; and there are many facts suggestive of hysteria. The character of the spasms, the clenching of the hands, the pronation and extension of the arms, the rigidity of the back, and the rolling about of the head, are all suggestive of hysteria. Then the patient feels a choking sensation. The globus hystericus is a very suggestive symptom. There is no optic neuritis. That is a fact of the greatest diagnostic value. The diagnosis of hysteria is confirmed by the previous history. The severe illness which the patient had, ten or eleven years ago, was obviously hysteria.

The differential diagnosis of hysteria and cerebral tumour is sometimes a very difficult matter. Dr Hughes Bennett has recorded a very remarkable case, in which a patient, who presented very marked symptoms of hysteria and who had no definite symptoms of organic disease, died from what was proved by post-mortem examination to be a cerebral tumour. I have myself met with cases in which there was the greatest difficulty in diagnosis.

It is of the greatest importance to remember that the mere presence of hysterical symptoms does not justify a diagnosis of hysteria—of mere hysteria, of hysteria only. Before you can diagnose mere hysteria, you must satisfy yourself that there is no organic disease.

Hysteria is a very common condition. In some women, hysterical symptoms are very easily produced by anything which lowers the nerve tone, the nervous control. A cerebral tumour, or any other organic nerve disease, may consequently be the cause of the hysteria. It is very important to remember this in practice. One should be cautious in committing oneself to a diagnosis of mere hysteria. A diagnosis of mere hysteria should never be given unless you have failed, after an exhaustive and careful examination, to exclude organic disease. Cases are every now and again met with in which it is very difficult or impossible to come to a positive conclusion between hysteria and cerebral tumour. Fortunately such cases are rare. A careful observer can, in the vast majority of cases, come to a definite opinion on the point. It is a matter of the greatest practical importance that a definite opinion should be arrived at. As I am in the habit of repeatedly telling you, it is difficult or impossible to cure a patient of hysteria so long as you are in doubt as to the true nature of the symptoms. The fundamental requirement for the successful treatment of hysteria, is to impress the patient with the belief that she will get better, that there is no organic disease, that you can and *will* cure her. Hysterical patients are usually very sharp. They very soon see if you are in doubt as to your diagnosis. If they think you have any doubt as to the nature of the disease, they will almost certainly conclude that they are suffering from some serious ailment. Such a conclusion will almost certainly retard or prevent recovery. In order to cure them, you must impress them with the belief that you *can* and *will* cure them.

In this particular case, the diagnosis is, I think, sufficiently clear to warrant a definite opinion. I will assure the patient that there is no serious disease, and that there is no reason why she should not soon get well. We will give her a prescription for some hydrobromic acid and gentian. In many cases of hysteria, the exact nature of the drug which you give is a matter of secondary moment. What you want to do is, to impress the patient with the belief that the medicine *will* do her good, and that she *must* take the medicine regularly; and that if she *does* take it regularly, she *will* find that it will do her good. Each dose of the medicine, even if it is merely coloured water—provided only that she takes it with the firm belief that it *is* something which *will* do her good—recalls to her remembrance the favourable opinion which you have impressed upon her. On some future occasion, I shall refer in more detail

to this very important subject—the subject of mental therapeutics, as I term it. Time does not allow me to go into further details now. In addition to the medicine, we will advise the application of repeated blisters over the back of the head, where she feels the chief pain. If these measures do not succeed, the Weir Mitchell plan of treatment—isolation, massage, electricity, and feeding—would probably be curative. Isolation and the faradic current are most valuable remedies in the treatment of many cases of hysteria.*

III.—The Process of Compensation in connection with Destuctive Lesions of the Motor Centres in the Brain.

In the case of destructive lesions of the cerebral motor centres, the result (that is, whether compensation will or will not take place) seems to depend largely upon the fact whether the movements, which are represented in the centre which is destroyed, are in the habit of being performed bilaterally or unilaterally.

The movements of the two eyes, of the two sides of the face, chest, and trunk are, for the most part, performed in concert. We laugh, for example, with both sides of our faces, and although some persons are able, by means of a voluntary effort, to put each individual muscle of expression into separate and independent action, for most of us this is impossible without long practice. Some people even find it difficult to wink with one eye alone.

In short, there is reason to suppose that, in the human subject, those muscles on the two sides of the body, which are in the habit of acting in true association or concert, may be thrown into action from either hemisphere. Further information is, however, required before this supposition can be definitely accepted. Horsley and Beevor, in some recent experiments on the lower animals (dogs, monkeys, &c.), were unable to produce bilateral movements by stimulating any particular cortical centre on one side of the motor area of the brain.

Possibly this associated bilateral action, if it actually exists, is effected, as Broadbent has ingeniously theorised, by means of commissural fibres connecting the nerve cells of the trophic nerve nuclei in the pons Varolii, medulla oblongata, and spinal cord. (See Fig. 101.) But be that as it may, there seems to be sufficient grounds for supposing that when a cortical centre, which innervates muscles, which are in the habit of acting in concert with corresponding muscles on the opposite side of the body, is destroyed, compensation is effected and permanent paralysis prevented by the function of the destroyed centre being taken up and carried on by the corresponding centre in the opposite hemisphere of the brain.

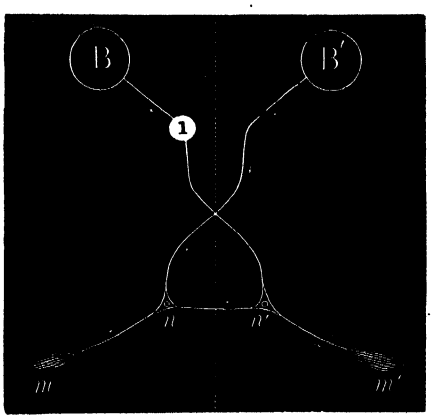


FIG. 101.—Diagram illustrative of the manner in which paralysis is compensated in the case of muscles which are in the habit of acting in association. The letter B points to the right, and B' to the left, cerebral hemisphere; *n* and *n'* to the nerve nuclei in the spinal cord, for the muscles *m* and *m'*, which are in the habit of acting together. A lesion, in the position of 1, will arrest the motor nerve force, passing down from B to *m* through *n'*; and will therefore cause paralysis of *m'*. Compensation is effected, and the paralysis recovered from by the motor nerve force from B', passing through *n* and *n'* to *m'*, as shewn by the arrow.

* Some additional remarks were made on the subject of "fasting girls." Want of space necessitates their omission from the report.

Studies in Clinical Medicine.

FRIDAY, FEBRUARY 21, 1890.

CONTENTS.

I.—THE PROCESS OF COMPENSATION IN CONNECTION WITH DESTRUCTIVE LESIONS OF THE MOTOR CENTRES IN THE BRAIN.

(*Continued from page* 296.)

IN the human subject, the more highly specialised movements, on the other hand, seem, so far as we at present know, to be represented only in one—the opposite—cerebral hemisphere. If this is so, we should expect that complete destruction of a cortical centre, say, for the muscles of the forearm and hand, would be followed by permanent paralysis; and that such is actually the case seems proved by the experiments of Ferrier, Horsley and Schäfer, in the monkey, and by the results of disease (destroying lesions) in the human subject. In those cases, for example, in which hemiplegia results from a lesion in early life, though the leg regains a considerable amount of power—indeed, in some cases, almost completely recovers, the muscles of the hand and forearm may, and often do, remain markedly paralysed. Such cases seem to prove conclusively that the motor centres for the muscles of the hand and forearm of one side are only represented in one, the opposite, hemisphere; and that when the muscles concerned in the production of the highly specialised movements of the hand are paralysed, compensation cannot be effected by the corresponding centre on the opposite side of the brain taking up and carrying on the function of that which is destroyed.

Cases are occasionally met with which seem at first sight, at all events, to form exceptions to this law. The most remarkable case of this kind with which I am acquainted is that which I have recorded in the 16th number of these *Studies*. In it, as the reader will remember, a large sarcomatous tumour, almost the size of the closed fist, had apparently destroyed the greater part of the motor area on the right side of the brain. So far as one could judge with the naked eye, the whole of the motor centres for the face and upper extremity were destroyed; and on microscopical examination, the grey matter in this region seemed to have completely disappeared. And yet there was absolutely no trace of paralysis.

Cases such as this suggest at all events the possibility of substitution being in some rare cases effected, even when the movements paralysed are highly specialised and differentiated, by the corresponding motor centres in the opposite hemisphere.

A more probable explanation is, however, that which supposes that the grey matter in the affected area was not entirely destroyed. In trying to give an explanation of anomalous

and exceptional cases of this kind, it must be remembered that the different cortical centres are not sharply defined and separated one from the other, that to some extent at all events they run one into the other. Such an overlapping has been clearly demonstrated by Horsley and Schäfer in the case of those centres which are situated on the inner side of the cerebral hemisphere (that is, in the marginal convolution).

The importance of this overlapping, and the possibility of compensation being effected by undeveloped or embryonic nerve-cells in the immediate neighbourhood of a centre which has been destroyed, has been pointed out by Victor Horsley, *Lancet*, July 1884, p. 7.*

In order to illustrate this theory of substitution, and to show the important difference that there is between rapid and slow destruction of a motor centre, I am in the habit of making use of the following simile:—Suppose a peal of bells, each rung by a ringer especially trained to ring his own bell and that bell only (the bell-ringers representing the discharging motor centres in the cerebral cortex, the bell-ropes the conducting fibres of the pyramidal tract, and the bells the muscles); and suppose that one of the bell-ringers, or discharging centres, is suddenly disabled—say, killed or put *hors de combat* by an apoplectic seizure—paralysis of the bell, or muscle, which he and he only has been trained to ring, will necessarily result; but if instead of being suddenly destroyed, one of the ringers is gradually and slowly disabled by some chronic disease—say cancer of the stomach—he will be able to communicate his failing condition to his fellow ringers, some of whom may be able to establish lateral connections, and to train themselves to carry on the work of their disabled colleague.

* See also article on the Plan of Construction of the Motor Mechanisms in the Cortex of the Brain, *Studies*, No. 13, page 223.

II.—Exophthalmic Goitre.

(*Continued from page* 291.)

In addition to the four great primary symptoms of exophthalmic goitre (viz., increased frequency of the heart's action; enlargement of the thyroid; prominence of the eyeballs; and the tremor, which Charcot so especially emphasises), quite a number of other symptoms may be present.

These symptoms have been arranged by Charcot in the table (see page 283), in accordance with the particular system (integumentary, digestive, urinary, etc.) to which they belong. On investigation, however, it will be found that the primary cause of almost all the "secondary" symptoms of Graves' disease, is derangement of some part of the nerve apparatus. The functional disturbance which results from this (primary) nerve derangement is manifested externally as a (secondary) disturbance of the function of some particular organ. Even the diarrhœa, which is a rare, but in some cases a very striking, symptom of Graves' disease, does not depend upon any mere local derangement of the gastro-intestinal tract; it is essentially the result of perverted innervation, its characters show that it is a true nervous diarrhœa.

In surveying the "secondary" symptoms of Graves' disease, I do not propose to follow in regular sequence the order of the symptoms as set forth in the table which I have copied (with one or two additions) from Professor Charcot. The description of these symptoms will, I think, be facilitated if we regard the manner in which they are produced, rather than the special organ, part of the body, or "system" which they implicate.

I have already described two of the most common and important of the secondary symptoms (viz., Stellwag's sign and Von Graefe's sign) in connection with the exophthalmos. I have also mentioned the occasional occurrence of ophthalmoplegia externa, and of inability to converge and accommodate for near objects, and stated that a remarkable case of the

former (ophthalmoplegia externa) has been published by Bristowe, and that the latter condition (inability to converge and accommodate for near objects) has been described by Möbius.

To these conditions I need not further refer.

Quite a number of the secondary symptoms of Graves' disease appear to be due to the disturbance of the vasomotor nerve apparatus. Let me now refer to some of them in more detail.

One of the most frequent, and perhaps one of the most important from a diagnostic point of view, is *diminished electrical resistance in the skin.* M. Vigoroux, one of Professor Charcot's assistants, was, so far as I know, the first to direct attention to the presence of this condition in cases of exophthalmic goitre. He found that in many cases of Graves' disease, an electric current passes through the skin with greater facility than in health. His observations have been confirmed by Professor Charcot, Dr Wolfenden, Dr Shaw, and others.

Dr Wolfenden (quoted by Sajous) found that the average resistance to the passage of a galvanic current of moderate strength was from four to five thousand ohms, while in eight cases of exophthalmic goitre the resistance which the body (skin) offered to the passage of the same strength of current was only from five to seven hundred ohms. In two cases of Graves' disease which he examined, the resistance of the skin was actually as low as two hundred and three hundred ohms respectively. The measurements are, of course, made by means of an accurately graduated galvanometer or galvanoscope.

This remarkable diminution of the resistance which the skin offers, in cases of exophthalmic goitre, to the passage of electric currents, appears to be due to vasomotor alterations, in consequence of which the capillary vessels of the skin are dilated; the tissues of the skin are, as it were, bathed in fluid, and the resistance of the normally non-conducting epidermis is reduced to a minimum.

This diminished electrical resistance of the skin is not, however, pathognomonic; it is not peculiar to Graves' disease; it may occur in other conditions in which the same degree of capillary dilatation of the skin is present. I have myself, for example, observed a notable diminution of the resistance of the skin to the passage of electric currents in some cases of chronic alcoholic excess. I am unable to give any definite measurements; and I do not know whether such a very remarkable diminution of the electrical resistance of the skin is observed in this or in other conditions, as Dr Wolfenden has noted in cases of exophthalmic goitre.

In some cases of Graves' disease, *profuse sweating* is observed. During the attacks of sweating, the skin may feel warm as well as moist. In other cases, the patients complain of *sensations of warmth* and *flushings* on the surface of the body. Charcot states that, in some cases, the feeling of heat or warmth (the flushings), which the patient experiences, is not merely a subjective sensation. The sensation of heat may be associated with an actual increase of the body temperature, which can be demonstrated by the thermometer. The rise in temperature appears to be the result of nervous derangements; as a rule, it is not associated with any discoverable local inflammation or visceral complication; nor is it attended with any of the urinary or other alterations which are usually present in pyrexia. This rise in temperature is said to be more common at the menstrual periods. In rare cases, the elevation of temperature is associated with grave symptoms of cerebral disturbance.

In some cases of exophthalmic goitre, attacks of *diarrhœa* are observed.

In the patient whose case is taken as the text of these remarks, diarrhœa was a very prominent symptom; it presented the peculiar features described below.

The characters of this diarrhœa are peculiar. It is apt to occur in paroxysms or bursts; it usually develops suddenly, quite independently of any apparent gastric or intestinal irritation; persists for some days, and, according to Charcot, is not readily controlled by ordinary astringent remedies, such as laudanum and bismuth; and, after lasting for some days, it spontaneously and rapidly disappears. The motions are frequent, copious, and watery,

and unattended by any pain or colic. During the attacks of diarrhœa, the appetite usually remains unimpaired; it may even be voracious.

In the case of exophthalmic goitre described in the text, the motions were said to be very copious, very liquid, and of a pinkish colour. The patient did not think that the pink coloration was due to the presence of blood. The patient only came under my observation as an out-patient, and I regret that I had not the opportunity of investigating the exact character of the motions for myself. I am not aware that this pink coloration of the liquid discharged from the bowels has been previously observed.

As a result of the repeated discharge of copious liquid evacuations, the patient may become much debilitated; but, as I have already stated, the appetite usually remains unimpaired throughout the attack, which is, as a rule, unattended with any of the ordinary symptoms indicative of gastro-intestinal irritation (pain, foul tongue, &c.).

In one of Charcot's cases, the attacks of diarrhœa recurred at regular intervals. The diarrhœa, which developed suddenly and without any apparent cause, lasted for seven or eight days, and then suddenly disappeared; the patient then remained free from diarrhœa for two or three weeks; another attack of paroxysmal and critical diarrhœa then occurred; this was again followed by two or three weeks of normal intestinal evacuation; then another paroxysm of diarrhœa, and so on.

In most cases of Graves' disease, in which this peculiar form of diarrhœa has been observed, the attacks of diarrhœa have been much less frequent than in Charcot's case, to which I have just referred, and have occurred at irregular intervals, and without any well marked periodicity. In this respect, and in its obvious nervous origin, the critical diarrhœa of exophthalmic goitre closely resembles the well known gastric crises of locomotor ataxia. There is, however, one notable difference between the intestinal crises of Graves' disease and the gastric crises of locomotor ataxia; in the gastric crises of locomotor ataxia, pain is a prominent symptom; whereas in the intestinal crises of exophthalmic goitre, pain is entirely absent.

In its mode of origin, the diarrhœa of exophthalmic goitre is perhaps similar to the profuse sweatings which are observed in some cases of the disease. It seems probable that the diarrhœa, like the sweating, is the result of a vasomotor derangement.

It is to be observed that in some rudimentary or imperfectly developed cases of exophthalmic goitre, the diarrhœa may be of diagnostic value, just as the gastric crises are of diagnostic value in some obscure and undeveloped cases of locomotor ataxia.

Pigmentation of the skin is a notable feature in some cases of Graves' disease. Dr David Drummond has directed special attention to this point; and as the result of my own observation, I fully agree with him in thinking, that it is a condition which is much more frequently present in exophthalmic goitre than is usually allowed, and that it may be of considerable diagnostic importance.

In some cases, the pigmentary deposits are distributed in patches, a common situation being around and below the orbits; in others, the whole skin is more or less uniformly stained, the pigmentation being in such cases usually darkest in those parts of the body (such as the areolæ of the nipples, the genital organs, the sides of the axillæ, &c), where pigment is normally in most abundance. In at least two cases of Graves' disease in males, I have been particularly struck with the dirty brownish-yellow colour of the skin, especially the skin of the face. In one of these cases, the disease existed in its rudimentary form, the enlargement of the thyroid and the prominence of the eyeballs being absent; but the other characters of the disease (increased frequency of the heart's action, tremor, nervousness, &c.), were so marked, as to make the diagnosis, to my mind, absolutely certain. I am disposed to think that this dirty yellowish-brown pigmentation of the skin is in some cases of considerable diagnostic value.

Patches of vitiligo have been observed in some cases of exophthalmic goitre. In Addi-

son's disease, patches of vitiligo may be distributed, here and there, over the deeply (abnormally) pigmented skin; the same association (excess of pigment in some parts of the skin, and diminution of pigment in others) is probably present in some cases of exophthalmic goitre.

I have not myself observed the presence of urticaria in cases of Graves' disease. Charcot includes it in his list of secondary symptoms. Considering the profound vasomotor disturbances which are present in many cases of the disease, one can readily enough understand the occasional occurrence of an eruption of urticaria.

Polyuria, which is probably in some cases, at all events, the result of vasomotor derangements, is met with in some cases of exophthalmic goitre.

Albuminuria also occurs in some cases. In one of my cases, in which a considerable amount of albumen was present, it was unassociated with tube casts, and did not, so far as I am able to ascertain, depend upon permanent organic changes in the structure of the kidney. In the case to which I refer, there were none of the ordinary symptoms of Bright's disease—either acute or chronic.

Glycosuria has also occasionally been noted in cases of exophthalmic goitre. If, as seems probable, the lesion in cases of exophthalmic goitre is central, and situated in the medulla oblongata, the occasional occurrence of glycosuria (and I may perhaps also add of polyuria and albuminuria) is not difficult of explanation.

A dry cough, which does not appear to depend upon any discoverable alteration in the respiratory tract, is sometimes present. In the later stages of one case, I observed a very profuse discharge of a bronchial mucus. The copious frothy expectoration was abundantly tinged with blood, which was uniformly distributed through it. The patient, who was under my care in the Newcastle-on-Tyne Infirmary, died during the attack. Post-mortem examination failed to reveal any other pulmonary lesion than œdema of the lungs. I have always regarded the pulmonary œdema and the profuse bronchial secretion which were present in this case, as the result of vasomotor paralysis. The condition was, I believe, exactly comparable to the profuse sweating and copious diarrhœa to which I have already referred.

In many cases of Graves' disease, *psychical symptoms* are prominent. The most casual observer cannot fail to be struck with the general "nervousness" of the patients affected by the disease. The least thing "puts them into a tremble." Little things, which would be unnoticed by any ordinary individual, are sufficient to agitate and disturb them. And yet they are not, as a rule, hysterical. Charcot, indeed, points out that some patients, who prior to the onset of the disease were distinctly hysterical, cease to be so, when once the symptoms of Graves' disease have become fully developed; and that, with the decline of the disease and the disappearance of its characteristic symptoms, the hysterical manifestations again make their appearance.

In some cases of Graves' disease, mental alterations of a more decided kind are developed. In many cases, the patients are fidgety and markedly irritable; in some, a condition of mental depression or actual melancholia is produced; in others, the patient becomes excited or maniacal. A fatal termination is sometimes preceded, or accompanied, by the development of acute mania. Symptoms indicative of general paralysis of the insane have occasionally been observed.

In some cases, *insomnia* has been a troublesome symptom. *Migraine*, *angina pectoris* and other forms of *neuralgia*, *epilepsy*, and *chorea*, are occasionally developed in the course of the disease.

Charcot mentions the occurrence of *paraplegia*, which, he states, presents certain peculiarities which seem to show that it is peculiar to the disease. The paralysis is usually incomplete; but in one case, the paralysis in the lower extremities was complete and absolute, at the time of the patient's admission to hospital. The paralysed muscles were flaccid and moderately atrophied, the reflexes, both deep and superficial, were abolished,—the electrical re-

actions were normal; there were no fibrillary twitchings; sensibility was unimpared, and the bladder and rectum were unaffected. When the paralysis was incomplete, and the patient was able to move about (either with or without the aid of crutches), a sudden giving way of the legs, due to sudden and unexpected flexion of the knees, was apt to occur; this sudden failure of the legs seems to be a characteristic and peculiar feature of the condition. The paraplegia is not permanent, it seems to be functional; but in the cases in which it was observed there were none of the usual symptoms of hysteria. Charcot states that the absence of hysterical symptoms, and the characters of the paraplegia itself (the fact that there is no sensory disturbance, &c.) seem to show that the paralysis is not hysterical.

In many cases, the *general health* is comparatively little, if at all, affected, though general nervousness is, as has been already stated, usually a prominent symptom. In some cases, more especially in the later stages, there is loss of flesh, and a cachectic condition is developed. If I may judge from my own experience, loss of flesh, marked debility, and cachexia, are more apt to be developed in the early stages of the disease, when the patient is a male. I have seen marked debility, emaciation, and cachexia, develop quite early in two a-typical or rudimentary cases of the disease. In both cases the patients were males.

Although in some cases, an *inordinate appetite* for food (bulimia) or sudden fits of hunger are present; in others, there is loss of appetite. Loss of appetite, a distaste for food, complete anorexia are, in my experience, usually associated with the cachexia and emaciation, which are apt to be present in the later stages of some cases of Graves' disease.

Charcot states that *jaundice* is developed in some cases. I have not, so far as I remember, met with any case in which this symptom was developed. In Dr Croom's case of rudimentary or imperfectly developed exophthalmic goitre, to which I have previously referred, the motions were abnormally pale and devoid of bile. Sir William Jenner seemed to attach considerable importance to this condition as a sign of Graves' disease.

A short dry (? nervous) *cough*, which is unassociated with any physical signs indicative of laryngeal or bronchial catarrh, is present in some cases. *Increased frequency of respiration*, which doubtless depends in many cases upon anæmia or cardiac complications, but which in some cases is perhaps of nervous origin, is not uncommon.

Menstrual derangements, more especially irregularity of menstruation and amenorrhœa, are frequently present. In the male, *loss of sexual power*, or even complete impotence, may be present in the later stages of the disease.

Enlargement of the lymphatic glands has been noticed in some cases. The *spleen* is also in some cases said to be enlarged. Whether these alterations are merely accidental complications, or whether they are in some way or another, which we do not at present understand, related to the disease, does not seem clear.

In a few cases, but, as has been previously remarked, they are comparatively rare, *cardiac complications of an organic* kind (endocarditis and organic valvular lesions) are developed.

Asthenia; asystole; relative and *muscular incompetence*, at the mitral or tricuspid orifices, with *œdema of the feet*; and *pulmonary complications*, such as œdema of the lungs and bronchitis, are, I believe, in most cases, the cause of death. It is, I think, doubtful, whether many of the conditions which have just been mentioned should be regarded as complications of the disease. I am disposed to think that in many cases, at all events, they are directly due to the disease—the result of the same nerve derangements, developed in a more intense degree, which are the cause of the symptoms which have been described as characteristic of exophthalmic goitre.

The consideration of the clinical history of the disease has occupied more space than I had anticipated. I must, therefore, defer what I have to say respecting the pathology, diagnosis, prognosis, and treatment, until the next issue.

III.—The Treatment of Typhoid Fever.

(Continued from page 278.)

The next point in the treatment of Typhoid, which we have to consider, is the regulation of the temperature. This is a very important question. Almost all authorities are, I think, agreed that when the temperature, in a case of typhoid fever, becomes excessive, it should be reduced and moderated. But observers are by no means agreed as to whether the temperature should be restrained and moderated in all cases. Some indeed think, and the suggestion seems very plausible, that the pyrexia (elevation of temperature) may have its advantages, as well as its disadvantages, in such a disease as typhoid fever. They suppose that a continued high temperature is inimical to the duration of the germs which are thought to be the cause of this disease. They believe that the pyrexia has its advantages, in as much as it tends to destroy the germ poison, and consequently to limit the duration of the disease.

Now if that view is correct—if the presence of pyrexia is advantageous—antipyretic remedies are not only uncalled for, but theoretically, at all events, may be said to be contra-indicated. Theory, however, is one thing and practice is another. Even those authorities, who think that the pyrexia tends to limit the duration of the germ poison, and so to terminate the disease, are, so far as I am acquainted with their opinions, agreed that when the pyrexia exceeds a certain point, when the fever is very high (say 104° or 105° Fahr.), that it should be restrained and moderated. The continued duration of a high temperature—of high fever—is without doubt an exhausting and deleterious condition. We have seen that when the temperature remains high for several weeks together, as it does in typhoid fever, that the heart suffers—that it becomes soft and flabby, and that its muscular fibres undergo degenerative changes. Now this of itself is a real danger. But it is not only the heart which suffers. Probably all the tissues suffer in some degree. There can, I think, be little doubt that many of the nervous symptoms which are observed, in severe cases of typhoid fever, are due to changes in the nerve centres, the result of the pyrexia. It must, I think, be granted that the long continuance of high fever produces injurious results in all the tissues, and is attended with real danger. Now if this is so, it is clearly our duty to take measures to restrain and moderate the fever, more especially when it becomes excessive. Whether we believe that the fever process tends to limit the duration of the disease—to kill the germs which are the cause of the disease—or not, we must, I think, admit that when excessive and long continued it is distinctly injurious; and that it should be restrained by appropriate remedies.

Personally, I am prepared to go much further than this. Personally, I am disposed to agree with those who think that the administration of antipyretic remedies is useful and advisable whenever the temperature reaches or exceeds 102·5° Fahr. And the correctness of this position is, I think, amply confirmed by the results of the cold bathing plan of treatment, to which I will presently refer.

Now we may reduce fever in two chief ways:—either by the administration of those internal remedies, which we know by experience bring down the temperature; or by the application of cold to the surface of the body.

Both means have been largely used in the treatment of typhoid—and both have their strong advocates.

Two of the chief sources of danger in typhoid fever are failure of the action of the heart, and the severity of the nervous disturbances produced during the later stages of the attack. The nervous symptoms are probably, in part at least, due to the retention in the blood of urea and other excrementitious matters; but there can be no doubt that the long continuance of high fever is largely responsible for both of these conditions; as has been already pointed out, high fever, if long continued, is a direct cause of cardiac failure, while it may also produce profound nervous disturbance.

In selecting apyretic remedies for the treat-

ment of typhoid fever, we should endeavour to choose those which fulfil the following conditions:—

1st. Which reduce the pyrexia.

2nd. Which do not directly depress the heart's action.

3rd. Which do not interfere with the elimination of excrementitious matters by the kidney.

Some of the more important drug remedies which have been recommended as antipyretics in typhoid fever are:—quinine, salicin, salicylate of soda, salicylate of ammonia, antipyrin, antifebrin, salol, &c.

Salicylate of ammonium (in 10 to 20 grain doses) and antipyrin seem perhaps on the whole to be the best. Quinine is in some cases of typhoid fever a valuable remedy, but as an antipyretic it is certainly inferior to many other drugs. In order to reduce the temperature of a case of typhoid fever by means of quinine—to reduce it to any considerable and definite degree, I mean—very large doses (30 or 40 grains) are required; and in typhoid fever very large doses of quinine are apt to be prejudicial; they are apt, I think, to increase the diarrhœa and intestinal irritation.

Salicine, salicylate of soda (when pure), salicylate of bismuth, and salicylate of ammonium are valuable antipyretics. Many observers are, however, of opinion,—and so far as my experience goes I am inclined to agree with them—that salicylate of soda, at all events, is apt to produce depression.

Jaccoud (quoted in *Sajous' Medical Annual*, 1889, vol. i., H., page 63) emphasises the dangers of the employment of sulphate of quinia and of salicylate of sodium in the treatment of typhoid fever. He points out that among the different forms of delirium, which may occur in the course of typhoid fever, there is one which it is most important to recognise and avoid, and that is therapeutic delirium. This form of delirium may, he thinks, arise as the result of the administration of sulphate of quinia or salicylate of sodium in too large doses, or even in moderate doses, if too long continued.

Salicylate of bismuth, though so far as my experience enables me to judge a less powerful antipyretic than some of the other remedies which have been mentioned above, seems to be a valuable remedy in some cases of typhoid. It acts not only as an antipyretic, but as an astringent and antiseptic, soothing the irritated intestines and restraining the diarrhœa when excessive. It may be given in 10, 15 or 20 grain doses.

I have had no personal experience of this remedy in cases of typhoid fever, but I believe it to be a very valuable remedy in some other forms of diarrhœa—especially in some cases in which the starting-point of the diarrhœa has appeared to be a chill or exposure to cold (sitting upon a cold stone, etc.), or in which the intestinal evacuations are copious, liquid, and inclined to be fermented and frothy.

Salicylate of ammonium has lately been strongly recommended in the treatment of typhoid and other continued fevers in which antipyretics are necessitated. It is said to be much less depressing than salicylate of sodium. My own experience does not enable me to say whether this is so or not.

Antipyrin and antifebrin are undoubtedly some of the most powerful antipyretic drug remedies which we possess. If judiciously administered they are, I believe, of real value in the treatment of some cases of typhoid. The dose should be small—not sufficiently large to produce depression. Five grains of antipyrin repeated at frequent intervals, in accordance with the behaviour of the temperature and the other circumstances of the case, is, I believe, in some cases a very valuable plan of treatment. My friend Dr John Macdougal tells me that he has seen great advantage, in typhoid fever, from the administration of small (2 to 5 grains) doses of antipyrin together with small doses (3 drops by the mouth and 1 drop subcutaneously) of liquor strychniæ. The antipyrin given in this way seems to restrain the temperature, while the strychnine prevents any depression acting, and acts as a valuable tonic and stimulant to the heart.

The second, and by far the most effective method of rapidly reducing the temperature in

a case of continued fever such as typhoid, is the external application of cold to the surface of the body.

The patient may be immersed in a bath; or iced cloths, or cold compresses may be applied to the surface of the body (abdomen, etc.).

The cold bathing plan of treating typhoid has been very largely employed on the Continent—more especially in Germany—and is strongly recommended by a large number of able physicians. Personally, I have no experience of it, and I am unable to speak definitely and positively on the matter, but the statements which have been from time to time published in its favour seem to me to be very remarkable. From what I have heard I am disposed to think that it is a most valuable plan of treatment, and that its advantages are not by any means sufficiently recognised in this country.

There are several different methods of carrying out the cold-water treatment of typhoid, viz.: (1) *The cold bath*—the patient being at once plunged into cold water, of a temperature of 65° to 70° F.; and (2) *The graduated bath*, the temperature of which is from 90° to 100° F.; it is gradually cooled by the addition of cold water till the temperature reaches 65° or 70° F.

Glenard (quoted by *Sajous*) gives the following outline of the technique of the different methods:—

"There are three kinds of baths applicable to the treatment of typhoid fever—the full cold bath, the half bath with affusion, and the full warm bath gradually cooled. Each of these may be employed in accordance with special indications:—

"1. The full cold bath of a temperature of 18° to 20° C. (64·4° to 68° F.); and of the duration of fifteen minutes, into which the patient is plunged up to his neck. This bath is stimulating and refrigerant. The indication for its employment is found in a great majority of cases.

"2. The partial bath with affusion. The temperature of the bath is 28° C. (82·4° F.); duration from five to ten minutes. The patient is placed in it up to the nipples; the affusion consists in pouring upon the back and neck water of 10° C. (50° F.); after that he is briskly rubbed in the bath with a sponge or brush. This bath is stimulating, and is to be employed in typhoid fever of high temperature and where complications occur, specially the chest complications.

"3. The warm bath gradually cooled, the temperature to be between 5° and 6° C. (9° and 10·8° F.) higher than that of the patient; the duration from twenty to thirty minutes. During this interval, the bath is gradually cooled by the addition of ice or cold water, until the temperature at the conclusion of the administration falls to 20° C. (68° F.). If shivering occur, the patient is immediately taken out of the bath and placed in his bed, which has been previously warmed. This bath is refrigerant, but without stimulant effect. It is to be employed in cases in which there are affections of the heart, emphysema, &c.

"Experience has shown that unfavourable results are avoided when the cases come under treatment before the fifth day. It is desirable for a physician, who is not personally familiar with the action of cold water, in the treatment of typhoid fever, to commence by employing it in simple cases, especially in ones which come under treatment prior to the fifth day of the attack.

"The following plan is that invariably to be employed under such circumstances:—In a suspected case of typhoid fever, seen at the beginning, the choice lies between a large dose of calomel and the treatment by cold baths, the preference being given to the latter if the presumption of typhoid fever is strong. If the diagnosis of typhoid fever is probable, recourse should be had to the baths, whatever may be the symptoms. The full tub should be placed in the ward or chamber, parallel to the bed, at a distance of one or two metres, the floor properly protected by oilcloth, and a screen placed between the bed and the bath tub. A sufficient quantity of water should be used to cover the patient's body to the neck. It should be of a temperature of 18° to 20° C. (64·4° to 68° F.). The baths should be prepared without disturbance or noise. There should be placed on the floor, near the head of the full tub, two pitchers of cold water, of a temperature of 8° or 10° C. (46·4° to 50° F.), each containing four or five litres (quarts). A glass of water should be at hand. The first bath should be given preferably about four o'clock in the afternoon, unless there is some urgent reason for selecting a different hour, and the physician should be present. The rectal temperature is taken, the urine voided, and the patient is assisted into the full tub, the screen having been removed. If there is perspiration, the patient is dried before entering the bath. Cold water from the pitchers is poured upon the head and back of the neck from a height of eight or ten centimetres (thirty-nine inches) for one or two minutes, the amount being from two to three litres (quarts). Then a swallow of cold water or red wine is given. This being done, the whole surface of the body is briskly rubbed with a sponge or brush. The patient is made to rub his abdomen and chest. These frictions stimulate the peripheral circulation, prevent the accumulation of heat at any one point, moderate the sensation of cold, and help to pass the time; they are not indispensable. Shivering appears, as a general rule, between eight and twelve minutes; this is a necessary evil, to which too much attention is not to be paid. Toward the middle of the bath, or at its termination, cold water is again poured over the head and neck. The time occupied ought to be at least fifteen minutes, longer if the head is still warm and the cheeks red, or if the temperature of the patient has been very high before the bath.

"The patient should leave the bath without precipitation; he cannot take cold. Thoracic complications are caused by typhoid fever, and not by chilling. The air of the apartment should be pure and not too warm; the window should be opened in the intervals of the bath; during the bath it should be closed. On leaving the bath the patient should be lightly dried with a towel. The bed should be carefully made during each bath. If, on returning to the bed, shivering takes place, the limbs should be rubbed and a hot bottle placed at the foot of the bed. A cold compress, covered with oil-silk or flannel, should be placed over the abdomen, and a little warm nourishment administered.

"Three quarters of an hour after the bath, the rectal temperature should again be taken. The general prescription for the administration of the bath is as follows:—A bath of from fifteen to twenty minutes every three hours, day and night, as often as the rectal temperature, taken three hours after a bath, reaches or passes 36° C. (102·2° F.). During the bath, douche the head and neck three times, each time with two or three litres of water of a temperature of 10° C. 50° F.). A small quantity of nourishment is to be given (after each bath, that is to say, every three hours. Alimentation should consist of the following articles: milk diluted with coffee or tea or cocoa (a quarter of a litre each time); gruel or oatmeal, tapioca, or vermicelli thoroughly cooked; veal, mutton, or chicken broth, freed of fat when cold, and re-heated at the moment of administration. As a drink, pure cold water; the indication for wine or spirits is only urgent in cases that are subjected to this treatment late in their course. If the patient does not sleep, or sleeps badly, he is to have a draught of ice cold water, and the abdominal compress is to be changed every three-quarters of an hour. The rectal temperature should be taken and recorded every three hours, before the bath, and forty-five minutes after each bath. If the temperature before the bath is below 39° C. (102·2° F.), the bath may be deferred until it again reaches 39° C., the temperature being then taken every hour. If it is found, however, to be below 38·5° C. (101° F.), it is not necessary to take it again for three hours. The discharges from the bowels are to be preserved for inspection, and the total quantity of urine collected in the same vessel. Neither age, sex, menstruation, pregnancy, nor sweating (except that which occurs at the end of defervescence) in any way modifies the treatment. In women who are weaning their children, cold compresses must be frequently renewed upon the breasts. If diarrhœa persists, it is to be combated by cold compresses, which may be kept cold by the aid of a bladder of ice. If there is constipation, it is to be treated by cold enemata; and if this fails, by enemata consisting of cold water and one part of fresh ox-gall.

"When the temperature before the bath is very high, or the fall, forty-five minutes after the bath, is less than 1° C. (1·8° F.), the bath must be prolonged to eighteen or twenty minutes. It is rarely necessary to modify the general formula. When the temperature does not exceed 39° C. (102·2° F.) but yet reaches 38·5° C. (101° F.), it is necessary to treat this little exacerbation by baths of 20° C. (68° F.), and of five minutes' duration, in order to prevent the prolongation of the fever, or the occurrence of relapse, and to shorten convalescence. If a relapse occurs, it must be treated according to the general formula. When the temperature no longer exceeds 38·5° C. (101° F.), the defervescence is terminated, the baths are discontinued, and the patient ought to be treated as convalescent, but is to be kept in bed until the temperature has not at any time exceeded 38° C. (100·4° F.). He may then rise, and in a short time walk in the garden; he may prolong his promenades according to his strength, and one will be struck with the rapidity with which it augments with every outing. Proper precautions are to be taken against cold. As to alimentation, already, during defervescence there may be added to his soup, milk, or bouillon, either one or two raw eggs daily, or, a little later, one or two spoonfuls of scraped raw meat, or a little toasted bread or biscuit; but the aliment must always be given as liquids. During the treatment by baths, one attendant is required for the day and one for the night; these duties may be fulfilled by the members of the family. In a hospital, one bath tub may be made to do for a dozen patients, but it is better to use one for six. It is not necessary to renew the water every three hours; once in twenty-four hours is sufficient. The patient treated from the beginning in this manner never suffers from fæcal incontinence, and the rule is the patient should pass his water before entering the bath. In time of epidemic, the water of the bath, if it is not soiled, should serve for several patients, and should only be renewed two or three times a-day." *

IV.—Case in which Stellwag's Sign and Von Graefe's Sign (retraction of the upper lid, the result of spasmodic contraction of the fibres of Müller's muscle, and non-descent of the upper lid in looking downwards) were present, but in which there were none of the symptoms and signs of Exophthalmic Goitre.

Male; age 27; a labourer; June 26, 1889.

(The patient had not been seen before he came to the Clinic.)

Dr B. What do you complain of?

Patient. A sore head.

Dr B. How long have you had it?

Patient. Since Monday.

Dr B. What part of your head is sore?

Patient (placing his hand across the forehead). Here; it got into the left eye and it swelled.

Dr B. When was that? (The eye did not appear to be swollen.)

* Sajous' Annual of the Universal Medical Sciences, 1889, vol. I., II, page 45.

Patient. Last Monday.

Dr B. Had you the headache before that?

Patient. Yes, for a week.

Dr B. Have you suffered from anything else but the headache?

Patient. No.

Dr B. You have not been sick? You have not vomited?

Patient. No.

Dr B. Is your sight all right?

Patient. Yes.

Dr B. Are you a nervous man?

Patient. No.

Dr B. Do you ever suffer from palpitation?

Patient. No.

Dr B. Have your friends noticed any difference in your appearance lately?

Patient. No.

Dr B. They have not noticed anything wrong with your eyes?

Patient. No.

Dr B. Have you been subject to headaches?

Patient. No.

Dr B. Have you been previously strong and healthy?

Patient. Yes.

Dr B. (*to the Students*). I have asked this patient several questions which may appear to you to have little or no bearing upon the case. But some very remarkable symptoms are present, which some of you may have noticed. If you observe the eyes closely, you will see that every now and again the upper lid is retracted, evidently spasmodically retracted; and the sclerotic being exposed round the iris, the eye acquires during the contraction of the lid a peculiar staring look. The contraction of the lids is, you will notice, bilateral. Both are equally affected.

Now this symptom, retraction of the upper lid from spasmodic contraction of the involuntary muscular fibres of Müller, is seen in a considerable number of cases of Graves' disease, or exophthalmic goitre. It is known as Stellwag's symptom or sign. I do not think it is peculiar to Graves' disease. I have seen it in at least two cases in which there was no evidence of exophthalmic goitre, but in which the patients were suffering from symptoms of mental derangement. This man has not complained of any nervous symptoms except headache. He does not give one the impression of being in any way nervous.

Dr B. (*to the Patient*). Has your mind been in any way affected?

Patient. No.

Dr B. (*to the Students*). His eyeballs are not prominent. (After examining the heart and thyroid)—The heart's action is quite quiet; the pulse is of normal frequency; there is no enlargement of the thyroid. In fact, none of the symptoms indicative of Graves' disease or exophthalmic goitre are present. We can, I think, definitely and positively say that this is not a case of exophthalmic goitre; and yet it is a case in which Stellwag's sign is well developed.

In connection with the spasmodic retraction of the upper lid, which is so very marked in this case, it will be interesting to note whether Von Graefe's sign—another of the conditions which is observed in some cases of Graves' disease—is present.

The patient was here made to fix the finger and follow it downwards as it was moved from above the horizontal plane of his vision towards the ground.

The upper lid did not follow the movement of the eyeball; it remained retracted; was as it were left behind by the eyeball.

Dr B. (*to the Students*). Von Graefe's sign is remarkably well seen in this case. I have never seen it more marked.

You see therefore that in this patient two of the symptoms which are supposed (when present) to be highly characteristic of Graves' disease are unusually well marked, and yet the case is certainly not one of exophthalmic goitre. None of the ordinary symptoms indicative of that condition are present.

I do not know that any special significance can be attached to the presence of Stellwag's sign and Von Graefe's sign in this particular case. The patient has several bad teeth. Possibly the spasmodic retraction of Müller's muscle is reflex, and due to irritation connected

with these bad teeth. The exact cause of the headache is also doubtful.

(This patient was told to come back, so that the condition of the eyelids might be watched and more carefully studied. He, however, failed to do so.)

V.—The Differential Diagnosis of Hysteria and Intracranial Tumours.

Hysterical symptoms are not unfrequently met with in the course of cases of intracranial tumour, and, indeed, in all forms of organic cerebral disease; and in some cases, in which the symptoms and signs of hysteria are prominent, and the ordinary symptoms and signs of the organic lesion, with which the hysteria is associated, are ill developed, the differential diagnosis may be a matter of extreme difficulty. Indeed, in the earlier stages of some cases it may be impossible to come to a positive conclusion.

In dealing with cases of this description, it is essential to remember that the diagnosis of mere hysteria (nothing more than hysteria) is never justified unless the observer has, by the most careful examination, and by repeated observation, failed to detect any symptoms and signs indicative of organic disease.

The condition of the optic discs is without doubt the most important point in clearing up the diagnosis. If ophthalmoscopic examination shows the presence of double optic neuritis, the doubts and difficulties of the case are at once removed, for double optic neuritis is very rarely, if ever, associated with mere hysteria, but is very frequently, indeed generally, associated with cerebral tumours.

It will be observed that I do not say double optic neuritis is *never* associated with mere hysteria. For not only would such a dogmatic method of statement be, in my opinion, highly unscientific, but I am personally disposed to think would, as a matter of fact, be in this case absolutely erroneous. Double optic neuritis *is* sometimes met with in anæmia, in association with derangements of menstruation, in connection with errors of refraction, and other conditions,* in which there is no "coarse" intracranial lesion, and with which hysterical symptoms are not unfrequently associated. I am therefore prepared, sooner or later, to meet with a case of apparently mere hysteria, in which double optic neuritis is present. Further, we are still largely ignorant, on the one hand, of the exact pathology of hysteria and hysterical symptoms (hysterical paralysis, hysterical contracture, hysterical anæsthesia, &c.); and, on the other, the exact mode of production of the double optic neuritis which is associated with many intracranial lesions is still a matter of dispute. Now, in the presence of such ignorance it would, in my opinion, be rash and unscientific to affirm that the same (?) functional alterations which produce hysterical paralysis, hysterical contracture, or hysterical anæsthesia, *cannot* possibly cause such vascular alterations in the fundus as will result in the production of some degree of œdema and swelling of the optic papilla. My own view, however, is that papillitis is not at all likely to be produced in this way. I am of opinion that the papillitis which is so frequently associated with cerebral tumours results, in most cases (in a way which I need not now describe), from increased intracranial pressure; and I see no grounds for adopting Dr Hughlings Jackson's theory, that the papillitis is the result of a vasomotor alteration (at the periphery) produced by the lesion in the centre (the intracranial tumour).

Personally, therefore, I am disposed to think that the (?) functional changes which produce hysterical paralysis or hysterical contracture are not in the least likely to produce double

* The experience of any single observer is necessarily so limited, and even the knowledge which can be acquired by making one's self familiar with the accumulated experience of authorities is, when compared with the facts and possibilities of nature, so imperfect, that I question whether such terms as *never*, *cannot*, and the like, should ever be used in an unlimited and unqualified sense. It is, of course, quite another thing to use these terms in a limited and qualified sense, and to say, *so far as I know*, or *so far as is known*, such and such symptoms *never* occur or *cannot* occur in connection with such and such affections.

optic neuritis. But, while holding this view, I cannot, for the reasons given above, dogmatically assert that double optic neuritis is *never* associated with mere hysteria alone.

But be that as it may, double optic neuritis is so rarely, if ever, met with in connection with mere hysteria, and so frequently met with in cases of intracranial tumour, that its presence in a case in which the diagnosis lies between hysteria and cerebral tumour practically settles the question, and by the practical physician may be taken as a definite indication that the case is not one of mere hysteria alone.

And here I would briefly emphasise the importance of the use of the ophthalmoscope in the diagnosis of cerebral (and, indeed, of many other) affections. It is not too much to say that the ophthalmoscope is just as important in the diagnosis of some nervous affections as the stethoscope is in the diagnosis of many cardiac and respiratory diseases. The routine use of the one is just as necessary, if mistakes are to be avoided, as the routine use of the other; for just as some serious cardiac affections—aortic regurgitation, for example—may be unattended by symptoms, but are readily detected on stethoscopic examination, so the double optic neuritis, which is easily enough seen in most cases by means of the ophthalmoscope, may be unattended with any alteration of visual acuteness, or, indeed, any limitation even of the field of vision.

But while the ophthalmoscope enables us to clear up the diagnosis in those cases in which double optic neuritis is present, and to come to the practical conclusion that the symptoms are not merely due to hysteria, but that there is organic disease, it does not enable us to make a differential diagnosis in all cases.

It must be remembered that double optic neuritis is by no means always present in cases of intracranial tumour. When, then, the ophthalmoscopic examination is negative, we are still left in difficulty and doubt.

In cases of this description, the severity of the headache is a point of some importance. Purposeless vomiting, too, especially when it occurs first thing in the morning, or is apparently due to alterations in the position of the head, is a symptom highly suggestive, though not by any means certainly indicative, of organic disease. Localised epileptiform convulsions, or a distinct epileptic fit, are strongly suggestive of organic disease. Localised paralysis of the cranial nerves is of the greatest diagnostic value, for while paralysis of the limbs is not uncommon in connection with hysteria, paralysis of the face is rarely, if ever, observed even in hysterical hemiplegia; and isolated paralysis of muscles supplied by motor cranial nerves, such as the facial, is very rarely, if ever, hysterical.

In doubtful cases, a careful study of the distribution of the paralysis, the state of the reflexes, and, indeed, of the whole nervous system, together with a careful consideration of the progress and course of the case, will generally enable the physician to decide whether the case is merely one of hysteria, or whether, in addition to the hysterical symptoms, there is not some underlying organic disease.

One of the most striking cases, which has yet been published, illustrative of the difficulty that there sometimes is in distinguishing hysteria and intracranial tumour, was reported a few years ago by Dr A. Hughes Bennett.

The case is of such interest and importance that I make no apology for quoting a large portion of Dr Bennett's report in full:—

CASE 1.—*Dr A. Hughes Bennett's Case of Cerebral Tumour, with Symptoms simulating Hysteria.*

The patient, a young lady, aged 16, precocious as regards the development both of body and mind, whose father, brother, and sister were remarkably neurotic in character, consulted Dr Bennett on 1st April 1876, complaining chiefly of blindness, imperfect hearing, and loss of power in the lower extremities. Her mental condition had always been peculiar; she had exhibited marked erotic tendencies; "on several occasions she had been detected in more than equivocal relations with men, and it is stated that she was at least on one occasion overheard making indecent overtures to her own brother. She had been expelled from a Grammar School in

Germany, the offence being so bad that her schoolmistress declined to give an explanation for her dismissal, and her parents, knowing her habits, did not press for a reason. Subsequently she went to a school at a fashionable watering place near London, where her conduct was characterised by general misconduct. At various times she had fits of laughing and crying, and irregular movements associated with a nervous attack. In October 1875, and while apparently in good health, she declared that she had become completely blind. As this occurred immediately after correction for mutinous conduct, and as she had got the reputation of being extremely cunning and wilful, the schoolmistress and others thought she was malingering. A medical man called in stated that he could find nothing the matter with her. Some few days afterwards she completely recovered her sight. This still further seemed to indicate that her alleged complaint was either intended or imaginary. She spent her Christmas holidays at home, accompanying her family to evening parties, &c. She was then seen by a London physician, who stated that she was hysterical, and who ordered strict moral control and cold baths. On returning to school, which it seems she disliked, she again stated that she had become perfectly blind, and in addition deaf. She continued for some weeks in this condition, when her hearing became completely restored, but the blindness remained. She was attended at different times by several practitioners, who agreed that the symptoms were of an emotional or imaginary nature, and that the patient was hysterical, and they ordered appropriate treatment. About February 1876, she stated that she very rapidly lost the power of her lower limbs, and that she could not walk. This, it was supposed, indicated her desire to avoid the daily walks which she disliked, and to have drives instead; with this also the deafness returned. In this state she continued till 1st April, when I saw her for the first time.

"The condition of the patient was then as follows:—A tall, full grown, highly developed, healthy-looking young woman. She stated she was quite blind in both eyes. A variety of methods were tried to test this, but without any definite results. The pupils were both widely dilated, and very sluggish in their movements. On ophthalmoscopic examination, with the exception of the vessels of both discs being very slightly congested, the appearances were normal. She also stated that she was completely deaf in the right ear and partially so in the left one, so that we had to talk loudly to be heard. I observed, however, that on some days she could hear perfectly well. Examination of the ear revealed nothing. She complained of slight weakness in the lower limbs, and that she could walk only with the greatest difficulty. On examining them they were found of good muscular development. When lying on her back motion and sensation seemed perfect, but at times there was hyperæsthesia of the skin of the legs. When she walked she staggered and moved about awkwardly, and stated she felt as if her legs gave way under her. There was no tenderness or abnormal condition of the spine. She stated that she suffered frequently from severe headaches in the occiput, which, however, were not constant, and lasted only a few hours at a time. The memory and intellect of the patient were in no way affected. She described all her symptoms with the greatest minuteness and exactitude. She had that manner of exaggerating her ailments so common in nervous persons. She cried out if her foot were touched, but if her attention were taken away from it, firm pressure could be made without causing her to wince. The pulse was 100. The extremities were cold and blue. Temperature normal. Tongue slightly furred. Bowels constipated. Appetite good. She had not menstruated since her illness commenced. Otherwise all the systems were normal. On subsequent examination there was abundant evidence that masturbation was practised to a very great extent.

"The diagnosis being uncertain, no active treatment was adopted. General hygienic and dietetic measures were suggested till the case had been further observed. The above conditions remaining, an eminent London physician saw the case with me. He came to the con-

clusion that it was one of aggravated hysteria, and gave as his opinion that little was to be done except placing her under judicious moral control. A respectable and experienced nurse was engaged to look after her under my direction, the patient remaining at home. There was no special medicinal treatment except occasional simple domestic remedies. She in no way improved. Every now and then she had attacks lasting from one to three hours, which had all the appearances of hysterical fits, involving shouting, laughing, crying, throwing herself about, striking the nurse, etc. On 15th April, her blindness remaining as before, she stated that her deafness had so increased that she could only hear with the greatest difficulty. The nurse said that at times she could hear quite well. She could now hardly walk, and could only with difficulty crawl from one room to another with assistance. On four or five occasions another physician * saw her with me, and after considering her condition from every point of view, he was of opinion that it was a case of hysteria. On 25th April she became restless and greatly excited, chiefly at night, and frequently alarmed the household by crying out and getting out of bed. Change of air and scene were judged desirable, so she was boarded in a private house in the suburbs of London. The excitement continued to increase, till on the 28th there were wandering and delusions. She was now totally blind, deaf, and could not stand or move her limbs. She did not know any one, and passed the contents of the rectum and bladder in bed. Her appetite failed; she complained of violent headache; her pulse was 140. Temperature could not be ascertained. On 1st May she became unconscious and wildly delirious, raving chiefly about some faults she had committed at school; about babies and their birth, tying the umbilical cord, &c. Pulse, 150. These symptoms continued till the 4th, the patient becoming gradually weaker. In the afternoon she appeared quieter; and at my visit, about 5 P.M., I found her seated in an armchair at the fire. It was obvious she was very feeble. Shortly after this she became suddenly comatose, and in spite of every effort to support her strength with nutrients and stimulants, she gradually sank at about 2.45 A.M. next morning.

"At the autopsy, which I had the greatest difficulty in obtaining, Dr Robert Lee kindly assisted me. We found the body well nourished, and all the organs healthy except the brain. On removing the calvarium, the pia mater was found deeply congested, and scattered over its surface were found five or six tubercular-looking bodies about the size of small millet seeds. The vessels on the surface were greatly engorged with blood. On removing the cerebral lobes in thin slices, a tumour was discovered in the right hemisphere. On further manipulation this mass was found to be about the size and shape of a hen's egg, of a dark translucent colour, and of nodulated appearance. It was of soft consistence, highly vascular, and on its surface were small extravasations of blood. It occupied the medullary substance of the middle lobe superior to the lateral ventricle, with the cavity of which it did not communicate, although it caused a marked bulging of its roof." *

Several cases have come under my own notice in which this same difficulty in diagnosis occurred. Two of the most striking are as follows:—

CASE 2.—*Organic Cerebral Disease, presumably a Tumour pressing upon the Motor Strands of the Internal Capsule, in which the difficulty of making a differential Diagnosis between "Coarse" Organic Disease and Functional Derangement was considerable.*

Miss A., æt. 38, was seen twice during the early part of 1885 with Dr Struthers, to whom I am indebted for the notes of the early history of the case.

Some five years before I saw her she began to complain of neuralgia affecting the upper and middle branches of the fifth nerve on the left side. This neuralgia, which varied much in its

* Who, like Dr Hughes Bennett himself, was one of the first authorities in Europe on nervous diseases.

* *Brain*, vol. i., p. 114.

severity, but never entirely left her for any length of time, was followed by pain in the left side of the head, and later by occasional pain in the left arm and left side of the neck. During the year 1882 the gait became reeling, and there was inability to stand with the eyes shut, and nausea. In 1883 clonic tremor on movement was noticed in the left arm, and subsequently, but to a much less extent, in the left leg. At this time the knee-jerk was exaggerated on both sides, but especially on the left, and ankle clonus could occasionally be elicited in the left leg. During the years 1883 and 1884 the headache gradually became more severe, with exacerbations which were accompanied by gastric pains, vomiting, and diarrhœa, these paroxysms lasting for a couple of days. The left arm and left leg were now distinctly weaker than the right, and the patient complained of pain when these parts were moved. On more than one occasion she tumbled, and was found lying on the floor of her room; but it was doubtful whether she was on these occasions unconscious or not. Six weeks before I saw her, she had had an attack of sciatica in the left leg, for which she had remained in bed. For eighteen months her voice had been weak and whispering, but was temporarily restored under emotion and excitement. The vocal cords were thin and pale, but acted symmetrically. Vision was stated to be impaired, but no changes in the fundus were detected on the single occasion on which ophthalmoscopic examination was permitted. The handwriting, until shortly before my visit, had been unaltered. The intellect was unaffected, but the patient, who was naturally peculiar, had gradually become most obstinate, self-willed, and intractable. It was with great difficulty that she could be persuaded to get out of bed, to wash herself, or to interest herself in her surroundings. The nurse stated that on one occasion she had noticed the face drawn to one side.

The result of my examination was simply to corroborate the account which has been given above. The optic discs were found to be quite healthy, and, so far as could be tested by rough examination (without a perimeter), there was no limitation of the visual field in any direction. The other special senses and the tactile sensibility of the skin seemed normal, but the patient made complaint of considerable pain when the left arm and the left leg were moved, more especially in the left hip-joint, which appeared, however, perfectly natural. Pain and tenderness on pressure were also complained of in the left side of the head. The patient was with difficulty persuaded to get out of bed, to stand, and walk across the room. All these actions were performed feebly, but there did not appear to be any localised leg paralysis. The knee-jerk on both sides was exaggerated, but ankle clonus could not be elicited. Movements of the left arm were attended with a marked rhythmical tremor, and were distinctly weak. The bladder and rectum were normal. The urine had on two occasions contained a small trace of sugar, but was now quite healthy. The mental condition of the patient was obviously most peculiar. She spoke in a low whisper, and it was with difficulty that she could be persuaded to answer the questions which were put to her. This appeared to be the result of obstinacy and mental perversion, and did not appear to be due to any lack of comprehension.

The pelvic and all the other organs were normal.

The patient continued in much the same condition for some months; then acute cerebral symptoms with high temperature suddenly developed, and death speedily followed.

A *post-mortem examination*, unfortunately, was not obtained, for the patient (who lived by herself, and was independent), anticipating our wishes in this respect, had expressly stated in her will that the body was not to be examined.

In the next case, in which the hysterical symptoms were apparently the first indications of commencing cerebral disease, a correct diagnosis was quite impossible, for there was absolutely no indication whatever of organic brain disease.

Studies in Clinical Medicine.

FRIDAY, MARCH 7, 1890.

CONTENTS.

I.—THE DIFFERENTIAL DIAGNOSIS OF HYSTERIA AND INTRACRANIAL TUMOUR.

(Continued from page 312).

CASE 3.—*Amputation of the Breast for Malignant Disease; Hysterical convulsions without any other Nervous Symptoms; subsequent slow development of Paralysis and Coma; Cancer of the Cerebral Dura Mater.*

The next case, illustrative of the differential diagnosis of hysteria and intracranial tumour, is one which I saw with Dr Keith a few years ago. The patient, a middle-aged lady, was suffering from convulsions. Eight days previously the breast had been removed for malignant disease (cancer); the progress of the wound was in every way satisfactory, but, on the evening of the seventh day, general convulsive twitchings of the head, trunk, and extremities had developed.

At the time of my visit, some twelve hours after the commencement of the spasms, the temperature was normal, and the pulse between 90 and 100. The patient was quite conscious, and, with the exception of the spasms, there were absolutely no other signs whatever of nerve disease. The attack had come on without any emotional excitement or other apparent cause. The spasms, which were very violent, and which had continued uninterruptedly for twelve hours, consisted of rhythmical to-and-fro jerkings, in an antero-posterior direction, of the head on the trunk, and of symmetrical clonic contractions of all four limbs and of the muscles of the back and abdomen. The patient was lying flat on her back, and the lower extremities were extended. On inquiry into the history, it was ascertained that the patient was naturally of a sober and unemotional temperament, and that she had never complained of headache, sickness, or other cerebral symptoms. She had, however, some two years previously, while staying in a Continental hotel, got up through the night, and alarmed her friends and the household by screaming; for this attack no cause was ascertained.

The diagnosis which was formed was that the convulsions were hysterical. The patient was firmly spoken to, and told that she could and must control the spasms; that if she did not do so, the wound would probably break open, and her recovery would be interrupted, or, perhaps, altogether prevented; and a mixture containing chloral hydrate and bromide of potassium was prescribed.

Under this treatment, the spasms quickly subsided; the patient made an uninterrupted and speedy recovery, and in the course of a few days went home apparently quite well.

Some weeks later I learned from Dr Skene Keith that paraplegia had developed. I naturally, though as the result proved erroneously, concluded that the paralysis, like the convulsions, was functional in character.

The subsequent course of the case and the

report of the post-mortem examination is copied from a letter, which Dr Keith received from the family physician, and which he kindly forwarded to me:—"Miss —— died comatose on the 18th inst. She gradually became more and more paralysed: one faculty after another—memory, language, deglutition, and, finally, respiration becoming more difficult. I requested and got a post-mortem. We only examined the spinal cord and the brain. In the cord (the lower half only was examined) there was no trace of any morbid appearance, nor in its coverings. In the cranial cavity — 1st, the membranes were adherent to each other, and the dura mater to the calvarium along the margins of the longitudinal sinus, for about two-thirds of the whole extent from behind forwards; 2nd, The substance of the brain was perfectly normal, and no appearance of disease in the membranes at the base was observable; 3rd, The adhesion of the membranes to each other at the part referred to, on minute examination, was found to be due to infiltration of warty-looking substance, which was found also in the choroid plexus in both lateral ventricles infiltrated in similar manner, and this substance showed under the microscope cells of multinuclear structure and of nested character in the meshes of the connective tissue. I have no doubt this had begun before the operation, and that the result was in no way affected by the operation."

II.—Exophthalmic Goitre.

(*Continued from page* 302.)

From the description of the clinical history of Graves' disease, which has been given in the two preceding numbers of these *Studies*, it is obvious that the symptomatology of the disease has, of recent years, been so fully elaborated that we may now be said to possess a very complete and accurate knowledge of the clinical features of the disease.

The pathology and the *pathological physiology* of the affection are in a very different position. I do not think that it is stating the case too forcibly to say that as yet we have little or no positive evidence as to the exact nature of the lesion which is the pathological substratum of exophthalmic goitre. Further, we are as yet uncertain as to the particular part of the nervous system which is primarily affected; though, as I shall presently point out, there are strong reasons for supposing that the disease is due to a "*central*" lesion, which is probably situated in the medulla oblongata. A series of carefully conducted and exhaustive post-mortem examinations, by skilled and trustworthy experts in pathological neurology, are urgently demanded in order to clear up the pathology of the disease. Unfortunately for pathology, but most fortunately for humanity and for our patients, the disease is not, as a rule, fatal, and the cases which do die, do not as a rule die in our large hospitals. It is comparatively seldom, therefore, that the thoroughly skilled observer has the opportunity of investigating cases of Graves' disease post-mortem, and of endeavouring to clear up the pathology of the disease by the aid of modern methods of histological research. It is eminently desirable therefore that, when an opportunity does occur, it should be seized with avidity and enthusiasm. The investigation which is required in cases of this description is a laborious one. The parts which more particularly demand attention are the medulla oblongata and pons Varolii, the spinal cord from its junction with the medulla oblongata to and including the upper part of the dorsal region (the so called cilio-spinal region of the cord), the gangliated cord of the sympathetic in the neck, and the cardiac nerves and cardiac ganglia. Now, to thoroughly and exhaustively examine, by the aid of modern histological methods, all these different parts, even in a single case, requires several weeks, at least, of continuous and laborious work. Few men in general practice, even if they have the necessary skill in the *technique* and methods, are able to devote the time which is required to such a research. But the general practitioner is able to afford

most valuable aid in the elucidation of this and many other obscure pathological questions. Many of us, who are enthusiastic about these matters, would gladly embrace the opportunity of making post-mortem examinations in private practice. I have myself repeatedly gone long distances in order to secure a post-mortem in a case in which I was interested; and although I am unable to devote as much time as I could a few years ago to work of this kind, I am most anxious, whenever my other engagements will permit of it, to embrace the opportunity of examining post-mortem such an obscure and interesting disease as exophthalmic goitre. If any of my readers, therefore, who are situated within a reasonable and get-at-able distance, will kindly give me the opportunity of making a post-mortem in typical cases of exophthalmic goitre, cerebro-spinal sclerosis, facial hemiatrophy, Friedreich's disease, pseudo-hypertrophic paralysis, bulbar paralysis, progressive muscular atrophy, &c., I shall be very much obliged to them.

With regard to the morbid appearances which have been found in the bodies of patients who have died of exophthalmic goitre, there is little to be said. The thyroid gland is, of course, enlarged; its vessels have usually been found dilated; in old standing cases—and most cases which die are usually of several years' duration—the connective tissue of the gland is notably increased, and the proper glandular structure more or less encroached upon and wasted.

In most cases, the heart has been less affected than one would have expected. In many cases, it is almost normal in size and weight; in some dilated; in others, to some extent, but not usually to any marked degree, hypertrophied. In a few cases, there is evidence of endocarditis, or chronic valvular disease.

The arteries throughout the body are said by some observers to be dilated; and in some cases, their coats have been described as thickened.

The orbital fat is usually increased in amount; and it is to this increase, and to the dilated condition of the vessels at the back of the orbit, that the exophthalmos is usually thought to be due.

The straight muscles of the eyeball have in some cases been affected with fatty degeneration.

The gangliated cord of the sympathetic in the neck, and especially the inferior cervical ganglion, have been said by some observers to be sclerosed or degenerated. Other and equally good observers have found the cervical sympathetic perfectly normal. The balance of evidence seems to me in favour of the latter view. It is, I think, probable that in those cases, in which the sympathetic in the neck was degenerated or sclerosed, the lesion was accidental, or, at all events, not the primary and true cause of the disease. Further and more detailed investigations, by recent histological methods, and by observers who have been accustomed to examine the sympathetic, in conditions both of health and disease, are required to clear up this point. As Dr Hale White has pointed out, and as I can myself affirm, for some few years ago I examined with great care the gangliated cord of the sympathetic both in the neck and in the abdomen in a large series of different cases, pigmentary and fatty changes in the nerve cells, atrophy of the nerve cells, infiltration with leucocytes, and a considerable degree of sclerosis of the ganglia, are of common occurrence, and are by no means peculiar to exophthalmic goitre or any special form of nervous disease. I do not mean to say that there is no disease in which degeneration or disease of the gangliated cord of the sympathetic does not play an important part. Quite the contrary. It is probable, I think, that future investigation will show that in many diseased conditions the gangliated cord of the sympathetic is implicated; and that in some of these, the lesion of the sympathetic is perhaps the essential and primary change. But, so far as I know, the relationship of clinical symptoms and conditions, with definite morbid lesions in the gangliated cord of the sympathetic, has not as yet been demonstrated. And in the particular disease with which I am at present dealing (Graves' disease), there are, as I will presently point out, reasons for supposing that the

gangliated cord of the sympathetic in the neck is not the primary seat of the lesion.

In a recent communication, Dr Hale White has described and figured the presence of certain morbid changes in the medulla oblongata in a case of exophthalmic goitre. His paper is a suggestive one, and, by directing the attention of subsequent observers to the condition of the medulla oblongata, will be of great value in the elucidation of the subject; but, without further corroboration, it may, I think, well be doubted if the changes which he describes were the true cause of the disease. I have more than once found pathological changes (recent hæmorrhages) apparently identical with those which he describes, in exactly the same parts of the medulla oblongata, in cases in which, so far as I know, none of the symptoms characteristic of Graves' disease were observed during the life of the patient. It is, I think, quite possible that the changes which were present in Dr Hale White's case of exophthalmic goitre were the result (of the vascular disturbances), rather than the primary cause of the disease.

The suggestion which Dr Hale White advanced in his paper, that the hæmorrhages, which were evidently recent, were the result of structural changes in the nerve tissue of the medulla, too delicate to be detected by our present methods of investigation, is a plausible theory, confirmation of which must depend upon future observations.

But, be that as it may, the balance of evidence, at present acquainted with it, seems to suggest that the lesion, whatever it is, which is the primary cause of exophthalmic goitre is situated in the medulla oblongata, rather than in the cervical sympathetic.

The old view, which has long held the field, that the disease is due to irritation of the sympathetic, is at first sight plausible.

Irritation of the sympathetic produces, as every one knows, acceleration of the action of the heart; but, as many observers have pointed out, it is difficult to suppose that a mere irritative lesion should be so persistent and so continuous, as the lesion of exophthalmic goitre undoubtedly must be.

Then again, though irritation of the sympathetic in the neck might produce the palpitation and accelerated action of the heart, which are such conspicuous features of the disease, if this is the true cause of this condition, other symptoms which are known to result from experimental irritation of the cervical sympathetic in the lower animals, such as dilatation of the pupil, ought to be present.

But, as has been already pointed out, the pupil is not usually (in fact only rarely and accidentally) dilated in cases of exophthalmic goitre.

Again, some of the symptoms, such for example as the dilatation of the blood vessels, which is said to be present in the disease, would appear to indicate a paralytic condition of the vasomotor nerves, rather than an irritative lesion; though Benedikt has suggested that the vascular dilatation may be due to an irritative lesion of the vaso-dilator nerves which run in the sympathetic. Friedreich, on the other hand, has theorised that a paralytic condition of the vasomotor nerves, by causing an increased flow of blood through the coronary arteries, and therefore to the tissues of the heart, may, by producing an increased excitability of the cardiac ganglia, be the cause of the increased cardiac action which is such a constant symptom of the disease.

The results of post-mortem investigation seem clearly to prove that, in many cases at all events, no obvious lesion is to be found in the cervical sympathetic.

Further, the widespread character of the symptoms in cases of Graves' disease undoubtedly shows that a localised lesion in the cervical sympathetic cannot be the cause of the condition; and so far as I know, it has not as yet been shown that the abdominal and other portions of the sympathetic nerve-apparatus are the seat of any definite lesion.

The widespread nature of the vasomotor changes is highly suggestive of a central lesion.

Ballet (quoted by Landolt) has suggested that* the disease is due to a paralytic lesion of one or

* Sajous' *Annual*, 1889, vol. iv., B., 131.

other of the nuclei of the pneumogastric nerve in the medulla oblongata.

Von Graefe's sign, which is perhaps due to defective co-ordination of the action of the muscles which produce descent of the eyeball and of the upper lid, and the fact that in some cases of the disease the movement of convergence is defective, and that in other cases paralysis of the straight muscles of the eyeball (ophthalmoplegia externa) has been observed, are also suggestive of the presence of a central lesion.

There is, too, some experimental evidence in favour of the view that the disease is due to a lesion of the medulla oblongata. Brown-Séquard, Filehne, and Benedikt all claim to have produced some of the symptoms of the disease by experimental lesions in the medulla oblongata of the lower animals. Filehne, indeed, found that in one case a lesion in the neighbourhood of the restiform body resulted in the production of the three great primary symptoms of the disease, viz., increased frequency of the heart's action, enlargement of the thyroid, and prominence of the eyeballs.

The theory which has been advanced by Möbius, that the primary cause of the disease is the lesion of the thyroid gland; and that in consequence of the structural and functional changes in the gland, secondary alterations in the nerve centres result, which are the cause of the numerous other derangements, has, not, so far as I know, been accepted by any other authority.

The *diagnosis* of exophthalmic goitre in typical and well-marked cases presents no difficulty. In cases of fully developed Graves' disease, the appearance of the patient is pathognomonic. It is only in the rudimentary and atypical *(fruste)* cases of the disease, in which there is neither enlargement of the thyroid nor prominence of the eyeballs, that any real difficulty in diagnosis should occur.

In ordinary goitre the enlargement of the thyroid is usually greater and harder than in Graves' disease; pulsation and thrills are not to be felt over the enlarged gland; while in ordinary goitre, increased frequency of the heart's action (without which it is impossible, as I have already stated, to diagnose exophthalmic goitre) prominence of the eyeballs, the characteristic tremor, and the numerous secondary symptoms, some of which are almost always present in every case of Graves' disease, are not observed, or, perhaps, to speak more accurately, are only accidentally present.

It must not be forgotten that when one lobe of the thyroid is very much enlarged (in ordinary goitre), it may, by pressing upon and irritating the nerves (sympathetic and vagus) in the neck, produce some of the symptoms which are characteristic of Graves' disease. Increased frequency of the heart's action, and it is said, prominence of the eyeball on the side on which the sympathetic is irritated, may be produced in this manner.

In such cases, the marked and hard enlargement of the thyroid; the absence of thrills and murmurs over the enlarged gland; the history of the case; the surroundings, perhaps, of the patient (for, as is well known, ordinary goitre is much more prevalent—endemic—in some districts than in others); the fact that the prominence of the eyeball would probably be unilateral (for it is not likely that the nerve cords, sympathetic and vagus, on both sides of the neck, would be pressed upon and involved); the presence of other distinctive signs of marked irritation of the sympathetic in the neck, more especially of marked dilatation of the pupil (a condition which, as has been already stated, is not present in Graves' disease); together with the absence of the other secondary symptoms, some of which are usually present in exophthalmic goitre, would enable a careful observer to come to a satisfactory conclusion as to the nature of the case.

The conditions which are most likely to be confounded with rudimentary and atypical cases of exophthalmic goitre are functional derangement and organic disease of the heart, and hysteria.

In all of these conditions, increased frequency of the heart's action, and palpitation, may be prominent symptoms.

But in Graves' disease, the increased fre-

quency of the heart's action is, with some exceptions, continuous and persistent, while in functional palpitation and hysteria, it is essentially paroxysmal and intermittent. The subjective sensations, which are associated with ordinary functional palpitation, are too, as a rule, much more pronounced than in Graves' disease.

In doubtful cases, the presence of the following symptoms should be diligently looked for:—the characteristic tremor; pigmentation of the skin, flushings and sweatings; diminished electrical resistance in the skin; nervous diarrhœa; and the other (less important and suggestive) secondary symptoms, some of which are usually present in the atypical and rudimentary forms of the disease.

The age and sex of the patient, the conditions which excite the palpitation, the duration of the symptoms, and the history of the case, the presence of other symptoms and signs of hysteria (for in Graves' disease the hysterical symptoms, even when present, usually disappear with the development of the disease), and notably—for this is a very important point—the effect of treatment, would probably also afford corroborative information as to the true nature of the case.

In cases of organic cardiac disease, in which the heart's action is persistently accelerated, physical signs indicative of a well marked organic lesion are almost certain to be present. A careful investigation into all the symptoms and signs, the history, and the whole circumstances of the case, will, in the great majority of cases, enable a judicially minded and accurate observer to distinguish the two conditions (Graves' disease and organic cardiac disease) without difficulty.

The only cases in which real difficulty is apt to occur, are those exceptional cases in which Graves' disease is complicated with organic cardiac disease. In cases of this description, the diagnosis can only, of course, be determined by detecting the presence or absence of the other (primary or secondary) symptoms which are characteristic of exophthalmic goitre.

The secondary symptoms, which are most important and suggestive, have already been detailed in speaking of the differential diagnosis of Graves' disease and functional palpitation.

The *prognosis* in cases of exophthalmic goitre has, up to the present time, been a very difficult and uncertain matter.

Experience has shown that the disease is rarely acute, either in its onset or in its termination (*i.e.*, that the symptoms are seldom suddenly developed, and that they rarely rapidly subside); that in most cases the course of the disease is chronic; that in some cases (Gowers, I think, estimates them at a fourth of the whole), the disease is completely cured; that in a much larger proportion of cases incomplete cure, with perhaps temporary remissions and intermissions, occurs, the disease lasting for several, and it may be for many years; and that in other cases (the exact proportion is difficult to arrive at and has not been accurately ascertained) the disease progresses from bad to worse and finally kills the patient. I have myself met with five cases in which death was directly due to the disease.

Until quite recently the effects of treatment have been anything but satisfactory. It would appear, however, that the electrical plan of treatment, to which I will presently refer, affords much better results, and that, in the future, our prognosis should consequently be more hopeful.

In trying to form an opinion as to the probable duration and course of any particular case of exophthalmic goitre, the following are the chief circumstances which should be taken into account:—The age and sex of the patient; the rapidity of development, and the length of time which the disease has lasted; the rapidity of the pulse; the severity of the general constitutional symptoms, more especially the presence or absence of emaciation, marasmus, and cachexia; the effects of treatment; and the presence or absence of complications.

Other things being equal, the fact that the patient is middle-aged or old is, I think, more unfavourable than if she were young. This is perhaps, in most cases, only another way of saying that the longer the disease has lasted—

in a severe form—the smaller is the chance of recovery or complete cure; for in most cases, in which the patient is middle-aged, the disease has already lasted many years before the case came under observation.

Cases are, however, every now and again met with in which the disease develops at or about the middle period of life (from 35 to 40). As I have already stated in speaking of the ætiology, exophthalmic goitre is, in my experience, apt to develop later in men than in women; and if I may judge from my own experience, the disease is, as a rule, more apt to be severe in men than in women; or to state the matter more accurately, the cases which occur in men are usually, I think, severe; whereas the cases which occur in women may be either mild, moderate, or severe. Men are much less frequently affected than women. In my experience, rudimentary and atypical cases are (proportionately to the number of males affected) more common in men than in women; and yet the affection is, I think, as a rule, more severe in men than in women. The rudimentary cases in men are, I think, more severe than the rudimentary cases, or even the average run of fully developed cases, in women. I would not, however, lay too much stress upon these points. They simply represent the impressions which I have gathered from my own experience; and the experience of any single observer is necessarily, in such a disease as this, far too limited to allow of any trustworthy generalisation.

The more rapidly the disease is developed, *in a severe form*, the worse, perhaps, is the prognosis. I am doubtful, however, whether very much importance can be attached to this point; it would appear that in some cases in which the symptoms of the disease have been quickly developed, they have also quickly disappeared or declined; in cases of this description, it is probable that the symptoms were not severe. I am disposed to think that when severe symptoms, cardiac and constitutional, are developed in the course of a short time, the case is likely to be a grave one.

The degree of the exophthalmos and the extent of the enlargement of the thyroid do not seem to afford any true criterion as to the severity of the case, the length of time it is likely to continue, etc. The frequency of the pulse is a much surer guide to the prognosis. Other things being equal, the greater the frequency of the pulse, the greater the severity of the case. *Vice versâ*, diminution in the frequency of the pulse rate is a favourable indication. In cases which recover, diminished frequency of the pulse rate is usually the first evidence of improvement. Slowing of the pulse rate is followed by diminution in the size of the thyroid and disappearance of the exophthalmos.

Pregnancy seems to exert a favourable influence upon the condition in some cases. Charcot emphasises this point, and quotes Trousseau to the same effect.

When the characteristic tremor is highly developed, the case is usually, I think, more severe than when there is no tremor. A high degree of general nervousness, profuse sweating, frequently recurring nervous diarrhœa, and pigmentation of the skin, are also, I think, indicative of the severity of the case.

But perhaps the conditions, which are most unfavourable from a prognostic point of view, are profond debility, emaciation, marasmus, and cachexia. Asystole, œdema of the feet, bronchitis, and œdema of the lungs, which are only seen towards the termination of bad cases, are, of course, still more unfavourable.

The manner in which the disease behaves under treatment is in Graves' disease, as in so many other affections, a point of the greatest prognostic importance,—and is so obvious that it need not be further insisted upon.

The presence or absence of complications must always, of course, be taken into account. It is impossible to go into details, the exact nature of the complication must of course guide the opinion in each particular case.

The *indications for treatment* are:—*firstly*, to remove all causes of excitement, whether of body or mind; *secondly*, to improve the condition of the general health; and *thirdly* to

employ such drug remedies, and other measures as experience has shown to be useful and salutary.

Persons suffering from exophthalmic goitre should be instructed to lead routine and quiet lives; everything which is found to accelerate the action of the heart, to produce palpitation, and to increase their general nervousness, should be strictly prohibited. It is needless to say that over exertion and anything which is likely to produce mental excitement should be diligently avoided and guarded against. In aggravated cases, and during the more acute stages, the patients should be confined to bed, or at any rate obliged to spend the greater part of their time in the recumbent position.

The diet should be easily digestible and nutritious. All articles of diet which are likely to produce flatulence or gastro-intestinal irritation should be prohibited. Tea, coffee, tobacco, and alcohol should be either prohibited altogether, or very sparingly indulged in.

A climate which is neither too cold nor too warm is perhaps the most suitable. A long sea voyage, provided the patient were not sea-sick, would probably be beneficial.

Stiller, quoted by Sajous (vol., iv. B, 131), reports a remarkable amelioration of the symptoms in two cases of exophthalmic goitre by the influence of high altitudes.

All causes of debility should if possible be removed. Any uterine or ovarian derangements which may be present should be attended to.

In those cases in which there is much anæmia, arsenic, or arsenic and iron, should be administered. The effect which iron produces on the patient should be carefully watched. Some observers have stated that it is apt to disagree, but I am satisfied that in some cases it is beneficial.

Arsenic, strychnine, quinine, the mineral acids, by improving the tone of the general health and of the nervous system in particular, are in some cases useful; but so far as my experience enables me to judge, they do not seem to exert any decided beneficial effect upon the diseased condition.

Of drug remedies, belladonna is perhaps, on the whole, the most useful. I believe that digitalis, or digitalis combined with iron, is in some cases beneficial. Some writers state that digitalis is contra-indicated in Graves' disease, but in the cases in which I have prescribed it, I have failed to observe any prejudicial effect. Brower, quoted by Sajous (*Annual of the Medical Sciences*, vol. iv. B, 131), states that he has cured several cases by the use of strophanthus; the dose was at first two drops of the tincture, gradually increased to ten drops. I have not as yet tried this remedy, and consequently have no personal experience to offer on the point. Aconite and the sulphate of sparteine have also been recommended; in the few cases in which I have tried these remedies, I have failed to observe any beneficial effect from their use. Ergot of rye has also been said to be beneficial.

Some years ago Charcot recommended the application of icebags to the præcordia; but lately he has abandoned this method of treatment in favour of electricity, which has been specially recommended by M. Vigoroux, and which has also yielded satisfactory results in the hands of numerous other physicians.

On the whole it would appear that the electrical plan of treatment is by far the most satisfactory which has yet been introduced.

Vigoroux and Charcot recommend that the constant current should be applied to the neck, and the interrupted current to the præcordial region.

In using the constant current, the electrodes should be firmly pressed deep into the neck, beneath the angle of the jaw on each side. The current may be allowed to pass from five to seven minutes at each sitting. Currents which are painful are too strong, and care should be taken that the current is not so strong as to vesicate the skin under the negative electrode. A current which, after it has been allowed to pass from five to seven minutes, gently reddens, but which does not vesicate the skin, and which is not painful to the patient, is, for practical purposes, the strength of current which is required.

The strength of the faradic current should be regulated by the sensations of the patient. The strongest interrupted current which can be comfortably borne may be employed.

In employing electricity in the treatment of exophthalmic goitre, it is well to remember that the patients are highly nervous and very easily agitated; it is therefore advisable to commence with weak currents. As the patient becomes accustomed to the treatment, the strength may be gradually increased, until the full strength of current, which it is desirable to employ, is used.

III.—Case of Sensory "Jacksonian" Epilepsy, in which a Discharging Lesion of the Half-Vision Centre in the Occipital Lobe produced Flashes of Light followed by Hemianopsia.

In previous numbers of these *Studies*, I have more than once referred to the localised convulsive seizures which result from an irritative lesion of the motor region of the brain; and which are usually termed attacks of "Jacksonian" epilepsy.

These localised epileptiform convulsions are of great importance from a localising point of view; for, by observing the muscles which are first convulsed, we are able to determine and localise the portion of the motor grey matter which is first discharged in the fit.

Two very important characteristics of a typical attack of localised epileptiform convulsions or "Jacksonian" epilepsy are the following:—*firstly*, there is no loss of consciousness, so long, at all events, as the spasms are localised and limited to particular groups of muscles; and *secondly*, the muscles, which are first and most convulsed in the attacks, are apt, in most instances at all events, to become temporarily paralysed; the paralysis is supposed to be due to the exhaustion of the motor grey matter which is discharged in the frequently recurring fits.

In a future number of these *Studies*, I will report a very striking case of "Jacksonian" epilepsy which came under my observation now many years ago, in which a small projecting spiculum of bone had produced a very localised but irritative lesion in the position of the motor centre for the platysma muscle.

My present object is not, however, to refer to the ordinary or motor form of "Jacksonian" epilepsy; but to state that irritative lesions of the sensory organs of the brain may produce symptoms which are absolutely identical in kind and sequence (the one, of course, being sensory and the other motor), with the symptoms which result from irritative lesions in the motor area.

These cases of *sensory* "Jacksonian" epilepsy are very interesting, and also of great scientific value, since they enable us to clear up, in a way which is difficult by any other means to do, the exact function of localised portions of the non-motor, or as they are sometimes, though, I think, erroneously termed, the non-excitable regions of the brain cortex.

In the following case an old syphilitic lesion, or cicatrix, adherent to the membranes on the one hand, and to the outer surface of the occipital lobe of the brain on the other, produced a series of recurring flakes of light, which were referred to the right eye, and right-sided homonymous hemianopsia. The case was first seen by me at the Edinburgh Royal Infirmary, on March 5th, 1887. The clinical details, which are given below, were published in the *Edinburgh Medical Journal* for August 1887, page 141. At a meeting of the Medico-Chirurgical Society at which the patient was shown, I suggested that the symptoms were probably due to a syphilitic cicatrix pressing upon and irritating some portion of the cortical centre for vision. I further stated that, because of the situation of the localised headache which the patient experienced during the attacks, and because of the peripheral constriction of the field of vision, which was present in the sound (seeing) halves of the retina, I was disposed to think that the lesion might probably be situated in the region of the

angular gyrus (the supposed higher centre for vision), rather than in the region of the occipital lobe (the part of the brain in which the half vision centre is located.)

The patient was carefully watched for some months after the full history of the case was published by my friend Dr John Thomson and myself, and I am glad to take this opportunity of thanking Dr Thomson for much valuable assistance, not only on this, but on many other occasions.

When death at last took place (the patient during the time that he was under our observation was affected with epithelioma of the tongue and adjacent parts) we were fortunate in meeting with no difficulty as regards the autopsy. On examining his brain, we found that the membranes were adherent over the outer surface of the posterior part of the left occipital lobe (see *a* in fig 102, which is erroneously numbered 98 in the plate) and over the adjacent surface of the cerebellum. The brain substance, for the depth of about a quarter of an inch underneath the position of the adherent membranes, was sclerosed and atrophied, and had evidently at one time been the seat of inflammatory changes. I have in my possession several microscopical preparations demonstrative of this point. But my present purpose is not so much to refer to the pathological as to the clinical aspects of the case.

I may state, however, before leaving the pathological side of the question, that this thickened patch of membranes presented no appearance of special interest. From the history of the case, and the rarity of localised cicatrices, other than those which result from traumatic injury and syphilis, I am strongly inclined to think that the cicatrix in this case was syphilitic. In other words, the pathological appearances corroborated the diagnosis made during life in regard to the presence of a lesion, and the nature of that lesion. Further, the lesion was, as I had expected, located in the visual area. It was situated, however, in the occipital region and not in the angular gyrus, as I had supposed.

The other parts of the brain, except those in immediate relationship with the cicatricial patch of adherent membranes, were absolutely healthy.

The case is a good example of *sensory* "Jacksonian" epilepsy.

It demonstrates that an irritative lesion on the outer surface of the posterior part of the occipital lobe may produce frequently recurring flashes of light in the corresponding halves of the retina, with which it (the half vision centre) is connected.

Further, the case proves that the irritative symptoms (the flashes of light) which result from the discharge of the half vision centre, are followed by, and may, while they are occurring, be associated with, a condition of temporary (exhaustive) paralysis of the elements which are discharged during the attacks. This temporary paralysis is manifested externally in the form of hemianopsia.

This case also shows that the half vision centre is located on the outer (as well as on the inner) surface of the occipital lobe. In most of the (few) cases which have yet been published, in which a lesion of the occipital lobe has produced hemianopsia, the lesion has involved the cuneus.

Further, the case shows that a localised lesion on the outer and inferior aspect of the occipital lobe, may produce loss of half vision of all kinds. In this case, the colour vision was equally affected with the other forms. This is a point of some little importance, for it has, if I remember right, been suggested that the half field for colours is situated in the upper (and not as this case would seem to show, in the inferior and outer) portion of the occipital lobe.

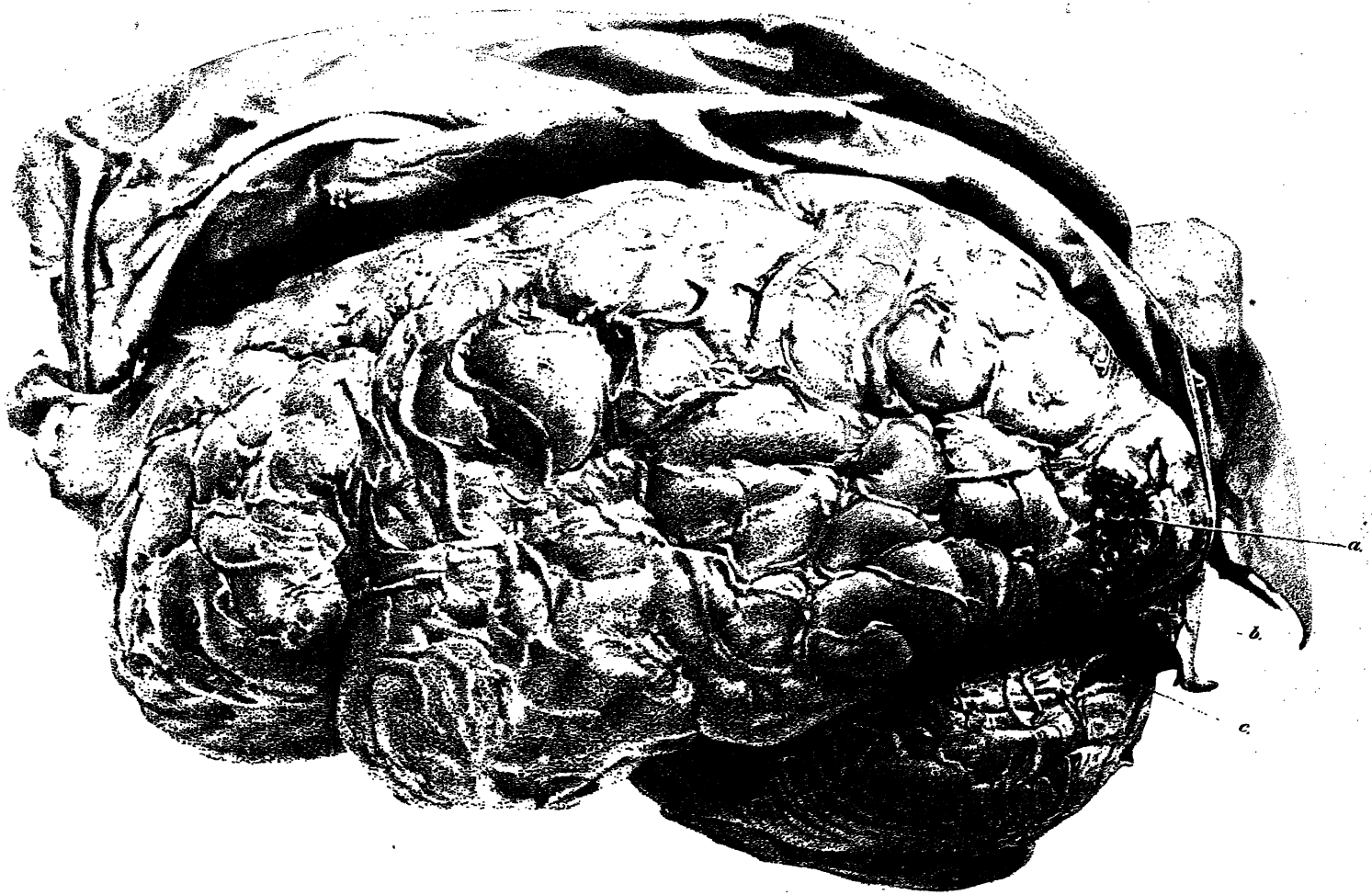
In the original report of the case, which was published in the *Edinburgh Medical Journal*, a series of chromo-lithograph charts, showing the fields of vision, both during the condition of paralysis (hemianopsia) and after recovery from it, were given.

The following is the report of the case:—*

Sensory "Jacksonian" Epilepsy; frequently Recurring Flashes of Red and White Light

* Copied from the *Edinburgh Medical Journal*, August 1887, page 141.

FIG. 98.—The outer surface of the left hemisphere of the brain, with the dura mater attached, in the case of sensory Jacksonian epilepsy, described in the text.



A thickened triangular portion of the membranes (to which the letter *b* points) was

referred by the Patient to the Right Eye; temporary Right-sided Homonymous Hemianopsia; the Condition being presumably due to an old Syphilitic (discharging) Lesion of some Part of the Cortical Centre for Vision on the Left Side.

Considering the frequency with which the ordinary (motor) form of "Jacksonian" epilepsy (localised epileptiform convulsions followed by paralysis of the muscles convulsed, the result of localised "discharging" lesions of the motor area of the cerebral cortex) is met with, it is not a little remarkable that so few cases of *sensory* "Jacksonian" epilepsy have been described.*

The rarity of sensory, as compared with motor "Jacksonian" epilepsy, may of course be due to the fact that localised irritative lesions, such as syphilitic deposits, much more frequently invade the motor than the sensory area of the cerebral cortex; but is probably, in part at least, if not altogether, accounted for by the fact that the effects of discharge and exhaustion of a motor centre (spasm and paralysis of muscles) are much more striking symptoms (both to the patient and to the physician) than the corresponding results of discharge and paralysis of a sensory centre. In other words, it is, I think, probable that cases of true *sensory* "Jacksonian" epilepsy are more common than is usually supposed.

It would be impossible to meet with a more typical case of sensory "Jacksonian" epilepsy, than the one which I am about to describe.

The patient, C. W., aged 63, an old soldier, who for the past seven or eight years has acted in the capacity of an usher in a large boys' school in Edinburgh, presented himself at my out-patient Clinic on 5th March 1887, complaining of frequent flashes of red and white light in the right eye. I may mention in passing, that Dr A. Hughes Bennett, who was present, was much interested in the case, and concurred in the opinion which was formed regarding it.

The patient, who is a very intelligent man, states that he contracted a chancre in 1848 (when he was 24 years of age), but that it was not followed by any secondary symptoms. The same year he was, during the Chartists' riots at Northampton, thrown head foremost through a shop window; the head and face were most severely cut, there being, he says, as many as 40 or 50 wounds—a statement which I can well believe, for the face and the scalp, which is bald, are now thickly covered with fine linear cicatrices, some of which are fully an inch in length. The bleeding which resulted from these injuries was excessive, and nearly proved fatal—a fact which is not to be wondered at when it is stated that the patient seems to be possessed in some degree of the "hæmorrhagic diathesis:" all his life he has noticed that slight cuts were followed by profuse bleeding; he has frequently suffered from severe epistaxis; and an operation which he recently went through—removal of a large portion of the left side of the tongue for epithelioma—nearly proved fatal by secondary hæmorrhage.

He enjoyed good health until the year 1863, when he came home from China. During that year he suffered, while stationed at Aldershot, from severe headache; the pain was chiefly felt over the back and top of the left side of the head; it was very much worse at night; he states that for two months he never got any sleep during the night, but was able to sleep a little during the day. The condition was termed by the doctors who attended him "chronic rheumatism of the brain." A large swelling, fully the size of half an egg, which was tender to the touch, developed on the head, at a point which he locates very exactly, about 1 or 1½ inches behind and slightly above the left parietal eminence. At this time there was also an extensive ulcer (the cicatrix of which

* In speaking of *sensory* "Jacksonian" epilepsy, I, of course, exclude, on the one hand, cases of ordinary epilepsy, in which the discharge begins in a sensory centre (say in some part of the visual centre), and is preceded by a sensory "aura" (say a red light); and, on the other, such conditions as "migraine," in which subjective visual sensations, such as flashes of light, indicative of discharge of gray matter, are often followed by very temporary defects of vision (hemianopsia) indicative of exhaustion or inhibition of the grey matter which was discharged.

still remains and is somewhat serpiginous in outline, but in no way pigmented) over the middle of the left calf.

During this attack of headache he had a severe epileptic fit, which was attended with loss of consciousness; the coma lasted for several hours. He has since had three other severe epileptic fits of a similar nature. The exact date of the second he does not remember. The third occurred in the year 1883, and the fourth in the year 1884. He does not think that the first and second fits were preceded by flashes of light in the right eye; but the third and fourth were.

For the past ten years he has suffered every now and again from "attacks of flashings" in the right eye. These attacks consist of a series of paroxysms. Each paroxysm lasts for a brief period (half a minute to two or three minutes). During the paroxysm he sees red and white flashes of light, exactly, he says, like a magic lantern. He states that the flashes are seen with the right eye. (The flashes are evidently seen on the right side of the middle line, and referred by the patient, as is usually the case in discharges of this description, to the eye on the side to which they are projected. The nervous elements discharged evidently correspond to the left half of each retina). The paroxysms occur with great frequency—every five or ten minutes—and the whole attack (consisting of innumerable separate paroxysms or discharges) lasts usually for three or four weeks.

At first these attacks used to occur about once a year—latterly, they have been more frequent, and have occurred every six or seven months. He knows no exciting cause for the attacks.

After the attacks have lasted for some time (usually some days), he has noticed that he is unable to see objects on his right side. He states that he is frequently knocked up against people, lamp-posts, etc., in the street in consequence of this blindness—a very "striking" fact in more senses than one. After the attacks of flashings ceased, the loss of vision (right-sided hemianopsia) also disappeared.

He has suffered more or less, but at irregular intervals, during the past ten years from headache. The pain is felt over the whole head, more especially on the left side, and is chiefly referred to a point on the back of the head, which is situated behind and above the left parietal eminence. There is no localised tenderness on skull percussion. There has been no vomiting, the optic discs are normal.

During these attacks of epilepsy of the visual centre, as they may be termed, he suffers more or less constantly from a noise in the right ear, resembling, he says, the noise made by a steam-engine; this noise is much worse during the individual paroxysms of flashings (*i.e.* epileptic paroxysms).

In 1886, an ulcer formed on the under surface of the left side of the tongue. For this condition he consulted Dr Alex. Miller, who admitted him to the Infirmary (in December 1886), diagnosed the condition as epithelioma, and removed the growth by operation. Severe secondary hæmorrhage followed the operation; it was restrained with the greatest difficulty, and nearly proved fatal. His speech is much impaired since the operation; and an ulcer has since formed on the inner surface of the gum on the left side, adjacent to the part of the tongue which was previously affected.

On examination, the patient, who is a very intelligent man, was found to be pale, but fairly well nourished. Several paroxysms of flashings were observed. During the attack, the eyeballs were very slightly turned to the right side; the palpebral fissures were slightly narrowed; very slight tremors were noticed in the orbicularis muscle; and the eyes had a somewhat vacant look, as if the patient was somewhat dazed, or was looking without fixation into space.

No perceptible difference was noticed in the condition of the pupil during the attacks. During the interval, the pupils were of medium size, somewhat sluggish in their action to light, but brisk in their contraction to accommodation.

The colour of the face and the character of the pulse were not perceptibly altered during the attacks.

At the first visit, the presence of right-sided homonymous hemianopsia was determined by the "rough" method of examination. The exact measurement of the field of vision, both for white and for colours, was subsequently mapped out with the greatest care by means of M'Hardy's self-registering perimeter (see perimeter charts in *Edinburgh Medical Journal*, August 1887, page 144).

The perimeter measurements showed complete right-sided homonymous hemianopsia, both for white and colours; and some peripheral constriction of the sound half fields.

The field for yellow was proportionately more contracted than the fields for blue and red.

Ophthalmoscopic examination failed to detect any marked abnormality in the fundus. There was no "word blindness." Smell, which for some years had been less acute in the *left* than in the right nostril, does not appear to have been altered during the present attack.

The heart, kidneys, and organs generally seemed to be healthy.

Progress of the Case.—Under iodide and bromide of potassium, the paroxysms of flashings soon subsided and the hemianopsia for white completely disappeared. The charts representing the fields, on June the 6th and 8th, (*i.e.* after recovery from the hemianopsia) were published in the original paper. On June 6th there still remained some slight peripheral contraction of the field for white in the right eye, more especially at the upper surface of the nasal half, while the area for white extended beyond the normal (as it did at the time of the hemianopsia) at the lower edge of the same (nasal) half of the field for that eye. The green field was also still very markedly restricted on the side of the former hemianopsia, and the yellow and blue fields to some extent contracted in the same direction.

In the left eye, the field for white was equal to the normal all round; it extended beyond the normal at the lower surface of the right half (the side of the former hemianopsia). The field for green in this eye was somewhat contracted towards the right.

On 15th June, the visual acuteness was found by Dr Lundie, who kindly examined the case for me, to be, after correction of refraction, normal.

The pupils, which were now very small, contracted actively both to light and to accommodation. The headache of which the patient formerly complained had disappeared.

IV.—The Treatment of Typhoid Fever.

(*Continued from page* 306.)

Dr Brand of Stettin, who introduced the cold bath method of treatment, insists that in order to get full benefit from it, the cold bathing must be commenced at the beginning of the attack and thoroughly and systematically carried out all through the disease. The temperature should never be allowed to rise above 102·2° F.; whenever it reaches this, not very high point, it should be again reduced by cold bathing.

Several years ago Dr Brand published the following very remarkable results of this treatment:—

In two German military hospitals, in which the cold bathing plan of treatment was thoroughly and systematically carried out, 400 cases of typhoid fever were treated without a death.

In two other German military hospitals, in which the treatment was employed, but in a less thorough and satisfactory way, the mortality was 3·5 per cent.

While in a number of other hospitals, in which the disease was treated without cold bathing, in the ordinary way, the mortality was 26 per cent.

More recently Brand has published the following results of his treatment:—

Cases treated in family practice— death rate,	-	0 to 1 per cent.
Military hospitals,		3 to 4 „
Consultation cases,		3 to 4 „
Civil hospitals,	-	5 „

In 1211 cases treated in private practice, and in the military hospitals, and the German Policlinic, *in all of which the cold bathing treatment was conducted systematically from the beginning of the disease*, the deaths only amounted to 1 per cent. Of the 1211 cases, only 12 were fatal; and two of these should be eliminated; for, in one of them, the treatment had to be discontinued on the sixteenth day on account of arthritis, and in the other, the fatal termination was apparently due to a relapse, the exact cause of death not being ascertained."*

"Drochon" (quoted by *Sajous*) has reviewed the statistics of various methods of treating typhoid fever. He finds that: 1. By former (ancient) methods the mortality is about 17.45 per cent., but Jaccoud, basing his estimate upon 80,149 cases collected from all sources, places the mortality at about 19.23 per cent. 2. The expectant method. Jaccoud, 11 per cent; Péter, about the same, 10.2 per cent.; in 1882–83, about the same; in 1882–83, he had 127 cases with 15 deaths, a mortality of 10.2 per cent. Dujardin-Beaumetz, 12 per cent; Bouchard, 15 per cent. Finally, Drochon has collected from the records of the Paris hospitals, 3,240 cases, with 405 deaths, or about 12.5 per cent. 3. The method of Brand, 6,185 cases, with 314 deaths, a mortality of 5.1 per cent. 4. Mixed methods. In 780 cases the mortality was 9 per cent.

Now, these statistics are certainly very remarkable. Statistics are proverbially unreliable; figures may doubtless be twisted and manipulated in various ways; and for the determination of many medical questions statistics are apt to be deceptive. But as regards this particular question, the percentage mortality which follows different plans of treating typhoid, I cannot myself see any reason for objecting to the statistical method—provided only that the figures are sufficiently large to eliminate any chance errors, and that the statistics have been compiled by trustworthy and reliable men. So far as I am able to judge, both of these conditions have been amply fulfilled in the particular statistics to which I am now referring. I cannot come to any other conclusion than this, that a comparison of the results of different methods of treating typhoid shows that the cold bath treatment, if systematically and thoroughly carried out according to Brand's directions, is attended with very striking results—with a wonderfully small mortality compared with other methods of treatment.

Many other observers, in addition to those already quoted, speak in the strongest possible terms of the advantages of the cold bathing method of treatment.

Richeaux, who has treated 76 cases by the ordinary methods, and 38 cases by the method of Brand, with a mortality in the first series of 10, and in the second of 5·25 per cent., says, "typhoid cases present an entirely different aspect since the methodical employment of cold baths. Medication by cold baths is not to be classed as part of ordinary treatment. It is a true method, applicable to almost all cases, and which produces in almost all positive advantages, and which is only rarely contra-indicated.

Wilson in summing up his admirable *resumé* of Glénard's article (in Sajous' *Annual of the Universal Medical Sciences*, to which I am indebted for so many of the foregoing statements), says, "Such is the treatment of typhoid fever by cold baths. When cases of typhoid fever are treated from the beginning by this method, the malady is as monotonous for the physician as for the patient. Neither the one nor the other has misgivings concerning the cure, which comes to pass without incident after a number of baths varying from sixty to one hundred, and after a duration of the fever of from seventeen to twenty-five or thirty days. If the duration be less than seventeen days, the fever must be regarded as having been a gastric fever. The advantages of the treatment consists in the reduction of the duration of the disease to its minimum, and in doing away with the convalescence, which is often so prolonged.

Dr Cayley, who has probably had a much larger experience of the cold bathing plan of treating typhoid fever than any one else in this country, speaks very favourably of it.

He seems, however, to think that relapses are

* (*Sajous*, 1889, vol. i, H. page 52.)

more common after it, than after the ordinary methods of treatment. At all events, this was the opinion which he expressed at a debate on the treatment of typhoid, which was held in London a few years ago.*

In the course of that debate, the opinion was expressed by more than one of the leading speakers, that the cold bathing plan of treatment is by no means free from danger. It was suggested that it is apt to produce pulmonary complications, such as collapse of the lung, and perhaps other grave results, such as sloughing and perforation of the intestine. The experience, however, of the German and French physicians, who have treated large numbers of cases by Brand's method, seems to have shown that such fears are groundless. Indeed, one of the advantages which is claimed for Brand's method is that it prevents pulmonary complications, perforation of the intestine, &c.

One of the chief objections which have been urged against the adoption of the cold bathing plan of treatment, as the routine and ordinary (everyday) method of treating typhoid fever, is the practical difficulty that there is of carrying it out, more especially in private practice. That this objection is not, however, a very serious one seems amply proved by the experience of Brand and numerous other continental observers. As a writer (whose name I am obliged to omit, for I have unfortunately mislaid the reference) has recently pointed out, it is perfectly certain that if the remarkable results which Brand and numerous other observers, who have thoroughly and methodically carried out this treatment, have obtained be accepted as accurate,—and, as has been already stated, for my own part I see no reason whatever for doubting their strict accuracy and truthfulness—the comparatively trifling difficulties of carrying out the cold bathing plan of treatment should not be allowed for a moment to stand in the way of its adoption, in private as well as in hospital practice. With a little experience, management, and determination, the practical difficulty of carrying out the cold bathing method will, in the case of better class patients at all events, be easily enough overcome. Provided that a couple of good nurses, who have had experience in Brand's method and are able to carry it out satisfactorily, can be procured—one for the night and another for the day—the practical difficulty of employing it in private practice will be reduced to a minimum. It cannot be doubted that, if the public once come thoroughly to realise and understand the enormous advantages which attend the cold bathing plan of treatment—the facts that under this method of treating typhoid fever, the mortality is reduced to one half, and the dangers of the disease reduced to a minimum—that it will insist upon being treated by Brand's method. One great objection, perhaps the greatest objection, which we have to face in trying to carry out the cold bathing plan of treatment in this country at the present time, is the prejudice which exists, both in the public and professional mind, against a mode of treatment which is so novel, and apparently so much opposed to our ordinary methods of dealing with the disease. Once this prejudice is removed—once the results which Brand and others, who advocate the adoption of this method as *the* ordinary, routine, everyday, plan of treating typhoid fever, are accepted as accurate—the practical difficulty of carrying out the method in private as well as in hospital practice will, to a large extent, disappear.

The results which Brand and his followers have published are so remarkable, that it is very desirable that those physicians in this country, who have the opportunity of treating a large number of cases of typhoid fever (hospital and workhouse physicians) should give the method a fair trial.

Before the method can be at all generally adopted in private practice, the profession requires to be satisfied as to its advantages. If it be granted that the results obtained by Brand and others are truthful and accurate, the method ought clearly to be adopted as *the* ordinary method of treating the disease. Once the method is adopted by the profession generally, there will be no difficulty in procuring a sufficient supply of capable

* The debate is reported in the *British Medical Journal* of November 27th, 1880, page 849.

nurses who have had experience of it. The demand has only to be made, and the supply of capable and experienced nurses able to carry out the treatment in all its details, will of course be forthcoming.

The next question to which I wish to refer in connection with the treatment of typhoid is the administration of stimulants. Until comparatively recently, alcoholic stimulants used to be freely given in most cases of the disease, but of recent years, the advisability of administering large quantities of alcohol as a matter of routine in cases of typhoid fever has been condemned by almost every authority. So far as I know, almost everybody now thinks that alcoholic stimulants should be reserved for the later period of the disease, and should only be given in large quantities in those cases in which there are special indications for their use. In the great majority of cases of typhoid, alcoholic stimulants are uncalled for during the earlier stages of the disease; it is only when the heart begins to fail and the pulse becomes markedly weak and dicrotic that alcohol should be administered. And in many cases of this kind it is perhaps doubtful if alcohol is the best form of stimulant which we possess. In many cases of typhoid fever—more especially in mild cases; in cases occurring in young healthy persons, possessed of a large amount of reserve force; and in cases which have been from the first methodically and systematically treated by cold bathing—alcoholic stimulants may be uncalled for through the whole course of the disease.

The very essence of modern methods of treating typhoid, and especially of the cold bathing plan of treatment, is to prevent the development of those conditions which call for the administration of alcohol. But while alcoholic stimulants are in many cases unnecessary, there can be no question whatever that their judicious administration is, in some cases, attended with the most beneficial results.

In treating typhoid, our object should be to prevent the development of those conditions which demand the administration of alcoholic stimulants; but, when those conditions do arise —when alcoholic stimulants do seem indicated —there should be no hesitation; brandy or some other form of alcohol should be freely given in accordance with the special requirements of the case.

In considering the advisability of administering alcohol in any particular case, we must take into account, the age and condition of the previous health of the patient; his previous habits as regards alcohol; the stage of the disease; and, especially, the condition of the heart and pulse, and the way in which the symptoms behave under the administration of the remedy, *i.e.*, whether the alcohol produces an obviously beneficial effect or not.

In young and previously healthy patients, alcohol may not be required throughout the whole course of the disease; whereas in older persons, and especially in those debilitated by previous disease, whose hearts are weak, whose vasomotor nerve tone is low, in those, in short, in which the stock of reserve force is small and speedily used up, abundant alcoholic stimulation may be very beneficial.

Persons who are accustomed to use alcohol freely, when in health, require, of course, to be more largely stimulated than those who are unaccustomed to its use. Those who are accustomed to use alcohol freely when in health, do not readily respond to alcohol, when attacked with fever. In order to produce any beneficial effect in such cases, the alcohol must be given in big doses, and frequently repeated.

The special indications for the administration of alcoholic stimulants are:—Great debility; a very rapid and feebly acting heart; a weak, small, and dicrotic pulse; symptoms embraced under the common term, the typhoid state; and the presence of pulmonary or other serious complications. During the debility of convalescence alcoholic stimulants are often also eminently beneficial.

Studies in Clinical Medicine.

FRIDAY, MARCH 21, 1890.

CONTENTS.

I.—THE TREATMENT OF TYPHOID FEVER.

(Continued from page 328.)

IN giving alcoholic stimulants in cases of typhoid it is very important, as I have already pointed out, to observe the effect which the remedy produces—whether that effect is distinctly beneficial or not. This is, I think, more especially necessary when alcoholic stimulants are given during the so-called typhoid state. I am satisfied that while in some cases in which profound nervous symptoms (the typhoid state) are prominent, alcohol is beneficial, in others it is injurious. Now, so far as I know, it is not easy or possible to lay down any very definite guides for the administration of alcohol in such cases—it is not easy or possible to say that in some cases, in which the patient passes into the so-called typhoid condition, such and such symptoms are present, and that then alcohol is indicated; whereas in other cases in which the patient passes into the so-called typhoid state, other symptoms are present which contra-indicate the administration of alcohol. So far as I know, the only satisfactory and really practical guide is, in such cases, the effect which the alcohol produces; and especially the effect which it produces upon the heart and pulse. If the condition of the heart and pulse and the general state of the patient improve, the alcohol should be repeated, and, if necessary, be administered in still larger doses.

Many different kinds of stimulants may of course be given in such a disease as typhoid fever; and in choosing the particular kind of stimulant which seems to be specially fitted for the circumstances of each particular case, we must of course have clear notions as to the effect which the stimulant is likely or calculated to produce.

As I have already more than once pointed out, the very essence of our modern method of treatment of typhoid, and especially of the cold bathing plan of treatment, is to moderate the pyrexia, and to prevent the exhaustion of the nervous energy, and of the cardiac force which the long continuance of high fever must necessarily produce.

One of the greatest dangers which we have to guard against in the treatment of typhoid fever is the failure of the action of the heart. All measures which prevent cardiac exhaustion, which preserve the reserve force of the nervous system and of the heart, which prevent the occurrence of hyaline and other degenerative changes in the cardiac muscle, are consequently valuable.

Strychnine is a very valuable cardiac tonic, and is eminently useful when given in small and frequently repeated doses in a long continued febrile disease, such as typhoid fever. I have already stated that my friend Dr John Macdougall, for whose opinion, experience, and judgment I entertain the very highest esteem and respect, has obtained very favourable re-

sults from the combined administration of strychnine and antifebrine. Digitalis and strophanthus may also, I believe, be given with advantage in some cases. The effect of digitalis, in particular, requires, however, to be carefully watched. Some authorities state that its administration is apt to be attended with injurious or even dangerous results, in cases in which the cardiac muscle is structurally degenerated, as it is in the later stage of a severe attack of typhoid fever. For my own part I feel satisfied that I have repeatedly seen injurious results from the administration of digitalis in cases of ordinary fatty heart (anæmic heart, true fatty degeneration, disease of the coronary arteries, &c.); but I feel equally sure that I have, in many instances, obtained very valuable therapeutic results from the administration of the drug in diseases such as acute croupous pneumonia and typhoid fever, in which, during the course, and as the result of a long continued process of pyrexia, failure of the action of the heart has arisen.

When sudden failure of the cardiac action occurs, and in those cases in which it is desirable to produce more rapid and energetic stimulation than can be obtained by the remedies which have just been mentioned, brandy, ammonia, and ether must be had recourse to. Musk is also recommended by many authorities.

With regard to the channel through which these remedies should be administered, one may give them either by the mouth or by the rectum. In typhoid fever, in which there is such a strong tendency to diarrhœa, and in which it is desirable to avoid every possible source of intestinal irritation, the former channel is preferable.

When still more rapid and immediate stimulation of the heart is required, the subcutaneous injection of ether, strychnine, or some of the other remedies which have been mentioned above, is a most valuable and important means of treatment.

The next and last point to which I wish to refer, is the treatment of some of the more important individual symptoms.

Diarrhœa requires to be restrained when it is excessive. A certain amount of diarrhœa is probably beneficial, since it helps to get rid of the intestinal sloughs. It is difficult to lay down any hard and fast rule as to what constitutes an excessive diarrhœa; much will, of course, depend upon the copiousness of the evacuations; and each case requires to be judged on its own merits. But, speaking generally, it may be said that, so long as there are not more than two or three or four motions, in the course of the twenty-four hours, special drug remedies, for the restraint of the diarrhœa, are not required.

In endeavouring to restrain the diarrhœa of typhoid fever, we have, as I have already mentioned, to pay particular attention to the dietary, and see that the diarrhœa is not due to the passage of undigested curds into the inflamed and ulcerated intestine.

Amongst drug remedies, opium and morphia are the most important. I am satisfied that I have repeatedly seen great benefit in typhoid fever from the administration of starch and opium enemata; but opium may be given by the mouth, or morphia may be administered subcutaneously.

Salicylate of bismuth, as has been previously mentioned, is a valuable remedy for the diarrhœa of typhoid. It probably acts not only as an astringent, but also as an antiseptic, exerting a cleansing action upon the sloughing and ulcerated gut. This is a point in the treatment of typhoid fever which is undoubtedly of considerable importance. There can, I think, be no question that some of the symptoms of the disease, and especially the irregular febrile disturbances—the ups and downs of temperature, which are in some cases attended with profound constitutional disturbance and sweating—are due to the absorption into the blood of poisonous (septic) materials from the ulcerated intestine. The administration of remedies, therefore, which prevent sepsis, and which tend to produce healing of the intestinal ulcers, is, more especially after the separation of the sloughs, likely to be beneficial. A drop of carbolic acid with two

drops of tincture of iodine given in a little simple syrup, or syrup of orange, every three or four hours is strongly recommended by some authorities (See *Sajous*, 1889, vol. i. H, page 62). Hydronaphthol, sulphocarbolate of sodium, or minute doses of corrosive sublimate, have been also recommended for the same purpose. Possibly menthol, given with a little castor oil, or dissolved in spirits of chloroform or tincture of cardamoms—a combination which I have found useful in some cases of flatulent dyspepsia and dilatation of the stomach—may be useful in the same way, but as yet I have had no actual experience of its effects. Washing out (lavage) of the rectum with pure cold water is recommended by some authorities. In some cases of typhoid, turpentine is useful. Ten drops given every three or four hours seems in some cases to exert a beneficial influence, not only upon the condition of the gut, but also upon the general state of the patient. When there is reason to suppose that the diarrhœa is due to the presence of curds or other irritating materials in the intestine, an occasional dose of castor oil, with or without a few drops of laudanum, is often very beneficial.

In those cases of typhoid in which there is *constipation*, the administration of castor oil or some other mild laxative is desirable. Some authorities recommend calomel in typhoid, but I have no personal experience of its effects.

Tympanites should be treated by the administration of opium or morphia, turpentine, and perhaps menthol given internally; by the application of turpentine stupes to the surface of the abdomen; or by drawing off the gas by means of a long tube introduced into the rectum. Dr T. J. Maclagan has recently published some cases in which the last named plan of treatment seems to have been beneficial (*Lancet*, March 8, 1890, page 537).

Hæmorrhage from the intestine should be met by the application of icebags or cold compresses to the abdomen, the subcutaneous injection of ergotine, the internal administration of opium and hazeline; and, where the hæmorrhage is profuse and death from collapse appears to be imminent, by the free administration of alcoholic or other stimulants.

Perforation of the intestine is an accident which is fortunately comparatively rare; but which, when it does occur, is almost uniformly fatal. Should perforation occur, probably the best thing we can do is to administer opium or morphia in as large doses as can be satisfactorily borne; to apply an icebag to the surface of the abdomen; and when the collapse is profound, to endeavour to prevent death by the administration of stimulants. Surgical interference has not, so far as I know, been attempted in such cases; and, looking at the conditions which are present, it does not seem probable that operative interference would be likely to be attended with success. The chances of unaided recovery from perforation in typhoid are, however, so exceedingly small, that any method of treatment, however desperate it may for *a priori* reasons appear to be, should not perhaps be absolutely condemned before it has had a chance.

There are several other points connected with the treatment of typhoid fever to which I had intended to refer, but the space which the subject has already occupied in the present volume of the *Studies* is so considerable, that I am reluctantly compelled to omit their consideration.

II.—Case of Epilepsy; Trephining; with Unsuccessful Result.

Boy; aged 8½; April 3, 1889.

This, gentlemen, is a case of some interest. The patient is a boy, an inmate of the Stirling County Asylum, whom I was asked to see a few weeks ago, on the recommendation of the Commissioners in Lunacy, with the object of advising whether the operation of trephining should be performed or not.

The patient has suffered from severe and frequently recurring epileptic fits for the past year and a-half. He was admitted to the Asylum in October 1888, on account of epileptic fits and mental derangement (dementia) associated with them.

It appears that when the boy was two years old, he fell off a table and severely injured the left side of the forehead; a large scar still remains at the seat of the injury, and the bone under the cicatrix is distinctly depressed. (The position of the scar is shown in fig. 103.) He was not rendered insensible by the fall; and according to his mother, he manifested no mental peculiarity until after the fits, from which he is now suffering, commenced.

He had his first fit in October 1887; and since that date he has continued to take fits almost daily. The fits are very severe; as a rule, he has several every night; and drug remedies, such as bromide of potassium, have failed to produce any beneficial effect upon his condition.

Soon after the fits commenced, a marked change was observed in his mental condition; and he is now almost completely demented. From the condition which he presents to-day, it is impossible to draw any accurate conclusion as to his usual mental condition. To-day he is suffering from the effects of large doses of bromide of potassium. When I saw him a fortnight ago at the Asylum, his appearance was altogether different. His expression and appearance were then remarkably bright and intelligent. (See fig. 104, which is copied from a photograph taken after his recovery from the operation. His appearance before the operation, and before he was dosed by the bromide, was quite as bright and intelligent as it is in fig. 104.) He was in fact quite unusually bright and intelligent-looking for a confirmed epileptic; and this peculiarity in the case is all the more remarkable since his mental condition is profoundly deteriorated. In fact, he seems to be completely demented, to be almost without any mind at all.

While in the Asylum at Larbert, he had been for a time actively treated with bromide of potassium, but without benefit; the drug had not, however, been given in very large doses; and it appeared to me that, before submitting him to operation, it would perhaps be well to give him larger doses of the bromide than he had previously taken. He has accordingly had twenty to thirty grain doses three times daily for several days. The fits have certainly not been quite so frequent, but they have still continued; the remedy has failed, even in these large doses, to restrain the condition. But while the effect on the epileptic condition has been comparatively trifling, the general effect has been most disastrous. You have only to look at the patient (see fig. 103, which was taken on the morning of his visit to the Clinic), to see that he is exceedingly ill. He is scarcely able to stand; his eyes are half closed; he can hardly be roused to take notice of anything; the saliva is dribbling from his mouth; and a gummy exudation is present at the angles of the orbits; for the past day or two he has scarcely eaten anything; there is a bromide rash on the surface of the body. The boy is, in fact, suffering from poisoning by bromide of potassium; and it would, in my opinion, be dangerous to continue the remedy any longer. It has certainly been pushed to its utmost limits, and that, you will observe, without restraining the epilepsy.

Under these circumstances, the question comes to be, whether anything more can be done; whether the operation of trephining is likely to relieve the condition or not. I have advised that the operation should be performed; and Mr Caird has kindly consented to admit the patient under his care, and to see whether surgical interference will produce any beneficial effect. I cannot, however, say, that I am sanguine as to the result. In this case, the operation may be said to be an experimental one. The presence of the well-marked cicatrix and the obvious depression of the bone, over the tip of the left frontal lobe, suggests the possibility that the epileptic condition may have its starting-point in the presence of a cicatrix or projecting spiculum of bone, which is irritating the surface of the brain.

FIG. 103.—The case of epilepsy and dementia described in the text. The drawing is copied from a photograph which was taken on the day of the patient's admission to hospital. The semi-comatose condition produced by the large doses of bromide of potassium is very apparent. The depressed cicatrix on the left side of the forehead is well seen.

FIG. 104.—The case of epilepsy and dementia described in the text. The photograph was taken after the operation. The semilunar cicatrix left by the operation is distinctly visible. The bright and intelligent expression of the patient, to which reference is made in the text, is well shown; it contrasts remarkably with the semi-comatose condition represented in Fig. 103.

This is a possibility, but there is nothing to show definitely that it is even a probability. The epilepsy did not show itself until five years after the injury. There is no tenderness on percussion over the region of the cicatrix. There has been neither headache nor vomiting; and there is no optic neuritis; in short, there are no symptoms and signs of a "coarse" intracranial lesion. The fits do not always commence in the same way, as one would expect them to do, if the epilepsy were due to a localised irritation starting in a particular part of the brain. In particular, it is important to note, that at the commencement of the attack, the head and eyeballs are not always turned to the same side; they are sometimes turned to the right and sometimes to the left; and the convulsive spasms are sometimes worse on the right and sometimes worse on the left side of the body.

The only symptom which may perhaps be a "localising" symptom, and which is suggestive of a lesion of the frontal lobe, is the profound mental deterioration (the dementia); one may of course have profound dementia after a long continued and severe epilepsy; but such profound dementia as is present in this case, is seldom produced so rapidly as it has been here. The case is also peculiar in as much as the profound dementia is associated with a remarkably bright and intelligent expression. Now, whether the mental aberration (the profound dementia), and the peculiarity to which I have just alluded are the result of a localised lesion in the frontal lobe or not, it is impossible, I think, to say. The dementia may be merely the result of the epilepsy; but it is suggestive of a local lesion in the frontal lobe.

Observers are agreed that the frontal lobes are the seat of the higher mental faculties, and lesions of the frontal lobe may undoubtedly be followed, in man, by marked mental impairment. It is quite possible, therefore, that the profound mental deterioration in this case may be the result of a lesion of the left frontal lobe.

This is, however, only a possibility, suggested rather by the presence of the cicatrix over the frontal bone at a point superficial to the anterior end of the left frontal lobe, than by the presence of other symptoms indicative of a local lesion in this part of the brain.

The anterior end of the frontal lobe, the præfrontal lobe as it is termed, is, as you are aware, non-motor. Electrical irritation of the anterior surface of the frontal lobe is not followed by localised muscular contractions,—but irritation of the posterior end of the upper frontal convolutions produces conjugate deviation of the head and eyes to the opposite side. Now, if the epilepsy in this case were the result of an irritation starting in the forepart of the frontal lobe, one would certainly expect that the spasm would commence always in the same manner, and that at the commencement of the spasm the head and eyes would always be turned to the opposite—the right side. Such is, however, not the fact. For this and the other reasons which I have already mentioned, I feel doubtful as to the presence of a lesion under the superficial cicatrix.

I have advised an operation, but as I have already said, it will be an experimental one.

The reasons which have induced me to advise an operation in this case are these:—The epilepsy is very severe; it has produced profound mental deterioration; drug remedies have failed to produce any beneficial effect; there is a distinct depression of the frontal bone at the seat of the previous injury; there may possibly be a lesion on the surface of the brain, beneath the external depression; and if there is such a lesion, it perhaps may be the cause of the epilepsy. At all events, it is, I consider, advisable to give the patient the benefit of the chance. I see no hope for him under any other plan of treatment, but, for the reasons which I have already given, I feel very doubtful if the operation will be attended with success or not.

Note on the subsequent course of the case.—The patient was admitted to the Infirmary under the care of Mr Caird. In the course of a week or ten days he had completely recovered from the

bromism. The skull was trephined at the region of the old injury. A marked depression on the outer surface of the frontal bone, to which the skin and scalp tissues were adherent, was found to be present. The inner surface of the bone was perfectly healthy. The external surface of the dura mater was healthy. The dura mater was opened; its inner surface, the subjacent soft membranes, and the portion of brain which was exposed, all appeared to be healthy.

The patient made an excellent recovery, and in the course of a few days was, so far as the effects of the operation were concerned, perfectly well.

The operation failed to produce any beneficial effect upon the fits. After a few weeks residence in hospital, the patient was sent back to the Stirling County Asylum. He has since, I understand, been transferred to the Asylum for Idiots at Larbert, his epilepsy and dementia being as bad as ever.

I append the report which I made upon the case; in some respects it is a little more detailed than the account of the case which has been given above:—

"I hereby certify, that on March 15, 1889, I visited the Stirling County Asylum, and in conjunction with Dr Maclaren, and his assistant Dr Bell, examined John M'Laren, aged eight and a-half years, who is suffering from epilepsy and epileptic dementia.

"From the particulars of the case which were furnished to me, it appears that the disease commenced in October 1887, when the boy was seven years of age.

"The first fit, which was a very severe one, seems to have come on without any obvious cause.

"Since October 1887, the fits have continued to recur with great severity and frequency. The intervals between successive fits was at the commencement of his illness about a fortnight; but since the patient's admission to the asylum (October 1888), the attacks have occurred almost every day—on an average at least five or six days in the week. There are now generally two or three, and sometimes as many as eight or nine, fits in the course of the twenty-four hours. The fits usually occur through the night, but not at any particular hour.

"Since the disease commenced, the patient's mental condition has undergone a marked deterioration; he is now practically demented and unable to speak sensibly. His mother states that, before the fits commenced, he was as intelligent as other children; but the correctness of this statement seems doubtful, for a neighbour informed Dr Bell that the boy was always rather peculiar, timid, slow in learning, and that he would often fail to pay attention to what was said to him.

"The family history is good. There seems, so far as can be ascertained, to be no tendency to any nervous complaint in any of his near relatives; in particular, none of his near relatives have suffered from epilepsy.

"His birth is stated to have been natural and easy.

"When two years of age, he fell from the kitchen table, striking the left side of his forehead against a chair. The injury was a severe one, and the wound had to be closed by sutures. He was not rendered unconscious by the fall. His friends did not notice any ill effects from the fall. He went to school like other boys, and according to his mother (see above) did not present any evidence of mental peculiarity or defect until the fits commenced.

"In October 1888, he became unmanageable at home, and was in consequence sent (on October 26th) to the asylum.

"The patient looks unusually bright and intelligent, considering the frequency and severity of the fits, and the very marked mental deterioration which is obviously present. So far as I could judge, he seemed to take little interest in his surroundings. I could not get him to answer any question which I put to him; and this it seems was not a temporary peculiarity, for Dr Bell, the assistant medical officer, stated that the patient can never be got to answer questions. He only made one sensible remark during the whole of my examination of him, viz., pointing to my hat, which was lying on the table near him, he said, 'What is that?'

"The patient is a well-grown and well-

nourished boy. There is no evidence of organic disease in any part of the body. In particular, there is no evidence of any 'coarse' intracranial lesion; the patient does not complain of headache; he does not suffer from vomiting; and there is no optic neuritis.

"On the left side of the forehead, immediately above the left frontal eminence, a very marked cicatrix, the result of the accident which he received when he was two years of age, and which has been previously described, is situated. A distinct depression of the bone can be felt at a point corresponding to the centre of the cicatrix.

"There are one or two small superficial scars on other parts of the surface of the scalp, but they are of no particular importance.

"Percussion of the skull at the seat of the cicatrix, on the forehead and elsewhere, does not elicit any complaint of pain or suffering.

"After an epileptic fit, the patient does not complain of any pain or uneasiness in the neighbourhood of the cicatrix.

"The epileptic fits do not appear to commence in any regular or definite manner. The patient does not complain of any definite warning sensation (aura). The spasms are sometimes more violent on the one (the right) and sometimes on the other (the left) side of the body. In particular, there seems to be no regular and constant deviation of the eyes and head to either side at the commencement of, or during, the convulsive seizures. The head and eyes are sometimes turned to one, and sometimes to the other side during the fit.

"The treatment which has hitherto been adopted, chiefly the internal administration of bromide of potassium, and the removal of a long and narrow prepuce by the operation of circumcision, does not appear to have produced any beneficial effect upon the fits. The convulsions continue to be as frequent as they were before, and there is little if any change for the better in the patient's mental condition.

"As the result of my examination of the patient, and consideration of the facts of the case, I am of opinion:—

"**1st.** That the case is a severe one, and that, so far as I can judge, the prospect of any marked improvement and of cure, as the result of drug treatment, is not hopeful. I believe, in short, that the patient will probably continue to be epileptic and demented, in spite of any drug treatment which may be adopted.

"2nd. That it is not improbable that beneath the depression, which can be distinctly felt from the outside on the left side of the frontal bone, there may be some deeper pathological alteration, such as adhesion of the dura mater to the surface of the brain, depression of the inner table of the skull, or an inwardly-projecting spiculum of bone.

"3rd. That if any such pathological condition is present, beneath the external depression, it is quite possible that it may be the cause (or perhaps, to speak more accurately, a cause) of the epileptic condition.

"I feel unable to speak more dogmatically and definitely on this important point, for there are no localising symptoms (unless the profound mental alteration be considered as such); and there is nothing to shew that the epileptic fits have their starting point in any particular part of the grey matter of this boy's brain. There are no definite indications (in the mode of the commencement of the spasms, the spread of the discharge, etc.) to show that the epileptic discharges originate in an irritation of the grey matter of the fore part of the first left frontal convolution, such as might be produced by the presence of a cicatrix on the surface of the brain beneath the external depression.

"The depression in the frontal bone appears to be situated over the upper surface of the anterior end of the first left frontal convolution—a portion of the brain which is non-motor and non-excitable.

"It is quite possible, therefore, that a lesion on the surface of this part of the brain might be present without any definite localising symptoms; and that such a lesion might in this particular case be the cause of the epileptic condition.

"The profound mental alteration which has so rapidly taken place since the fits commenced,

and the striking fact which must be regarded as a peculiar feature of this case, that while the boy looks bright and intelligent he is mentally so inert, may possibly be regarded as localising symptoms, and as suggestive of a lesion of the frontal lobe, which, there is every reason to suppose, is especially concerned with the higher mental faculties. At all events, these facts (the mental deterioration and the difference in the patient's appearance, and his actual mental condition) are quite in harmony with such a supposition (the presence of a supposed lesion of the frontal lobe).

"On the other hand, it must not be forgotten that electrical irritation of the back part of the frontal lobe is attended with conjugate deviation of the head and eyeballs to the opposite side; and I should certainly expect that in a case of epilepsy in which the discharge had its starting point in the grey matter of the frontal lobe, the head and eyeballs would at the commencement of, and throughout the attack, probably be turned towards the opposite side. In this particular case, no regular conjugate deviation to either side has been observed.

"To sum up the result of this somewhat long discussion:—The epileptic condition in this case may be the result of abnormal conditions, beneath the depression which can be felt on the exterior of the skull. I feel unable to say positively whether this is so or not. I see no prospect of material improvement under drug treatment. The operation of trephining, as now conducted, is not usually attended with danger to life. I would, therefore, advise that the operation be performed, that the bone be trephined at the seat of the depression, and that the dura mater be opened, in the hope of detecting and removing a cicatrix or other pathological condition which may perhaps be the cause of the epilepsy.

"EDINBURGH, *March* 21*st* 1889."

III.—CASE ILLUSTRATIVE OF THE MANNER IN WHICH MISUNDERSTANDINGS MAY ARISE BETWEEN MEDICAL MEN.

Married woman; aged 41; affected with progressive muscular atrophy.

THIS patient, gentlemen, came to the Clinic about six weeks ago. Many of you will no doubt remember the case. It is a very marked example of progressive muscular atrophy. The small muscles of the hand—the muscles of the ball of the thumb and the interossei—and some of the muscles of both forearms are notably wasted. There is loss of power in proportion to the loss of muscle; and both the muscular atrophy and the diminution of muscular strength have developed gradually and *pari passu*. As the muscular wasting has become more and more conspicuous, so has the loss of motor power become more and more apparent. There is absolutely no impairment of sensation. The patient's general health is good. The bladder and rectum are unaffected. One notable feature about the case is the fact, that the upper extremities, in which the atrophy is as yet alone apparent, are the seat of a fine tremor, which resembles the tremor of paralysis agitans more than anything else. It is quite different from the fibrillary twitching which can usually be observed in cases of progressive muscular atrophy. In this case, the tremor is a much more continuous tremor; it is not a mere intermittent twitch occurring at irregular intervals, every now and again. If you observe the thumb of the right hand, you will see that it is affected with a frequently recurring rhythmical movement, which reminds one of the movement of paralysis agitans. I need not, however, go into details either as regards the subject of progressive muscular atrophy generally, or the special features which this case presents. You will remember that I considered the subject pretty fully, when the patient first came under our notice, six weeks ago. There is, however, one point about the treatment to which I wish particularly to refer.

When the patient came before us six weeks ago, I prescribed a mixture containing arsenic and strychnine; and told her to continue to apply the electricity (the faradic current), which by the advice of her private medical attendant she had been using for some time.

Dr B. (to the Patient). Are you feeling better?

Patient. I feel a good deal stronger.

Dr B. Are your arms any better? Do you feel your arms any stronger?

Patient. They are much about the same.

Dr B. Is the trembling in the arms any less?

Patient. Yes; I think so.

Dr B. (to the Students). The muscular wasting is of course just the same. We could not expect to see any decided difference in that respect.

Dr B. (to the Patient). You must steadily persevere with the treatment—with the medicine and the battery. The disease from which you are suffering is very difficult to cure; and you must not be disappointed if you do not seem to improve as quickly as you would wish. It is only by going on steadily and perseveringly with the treatment for many months, that you can possibly hope to arrest the progress of the disease. If the disease progresses, it will prove to be very serious. By great attention to the treatment, and to the condition of your general health, you may hope to arrest the progress of the disease. But you must clearly understand, that even if the greatest improvement which we can expect takes place, it will be very slow in appearing. It is very important, too, that you should continue under the care of your family doctor. I shall be very glad to see you again in a few months' time; but it is not enough to come in here every few months; you want much more careful looking after than that. You must be guided by your family doctor as to whether the medicine should be increased or not, in the course of a few weeks; and you must be guided by him as to whether you should continue the electricity or not. Since you were last here, I have seen your doctor, and have had a talk with him about your case. He knows exactly what I think about you. I will write to him and tell him how I find you.

Before you go, there is one question I want to ask you. It seems that after you were here six weeks ago, your husband stated that you had been told that if you had come to the Infirmary two months sooner you would have been cured. Now did any one at the Infirmary tell you anything of the kind?

Patient. No.

Dr B. Was anything said by any one here which led you to suppose that if you had come for advice sooner, that you would have been more easily cured?

Patient. No.

Dr B. There was nothing said then which could have led your husband to make the statement which I understand he did make about your case?

Patient. No.

Dr B. I am glad to hear that. It is very unfortunate that he should have come to any such conclusion; or that he should have made any such statement. I entirely approve of the treatment which you were getting before you came here at first. I think I told you this when you were here before. Certainly I told you to continue the electricity and rubbing which your family doctor had prescribed. Now I want you to continue with this treatment; and as I have already told you, it is very important that you should see your doctor regularly and be guided by him as to the future treatment. You clearly understand that?

Patient. Yes.

Dr B. (to the Students, the patient having withdrawn). The case is a very good illustration of the difficulties of practice. It shows how easily misunderstandings between medical men may arise, and how essential it is not to attach too much importance to the mere statements of patients. Patients are very apt to draw absolutely erroneous deductions from statements which are made to them regarding the nature of their case; and to make all sorts of statements founded on their erroneous conclusions as to what a doctor says to them.

It seems that after this patient was here on the first occasion, her husband stated that she

had been told at the Infirmary that if she had come here two months sooner she would have been cured. That statement came to the ears of her medical attendant. He was naturally very much annoyed at it. But, being a sensible man, he did not attach any very great importance to it until it was corroborated. Fortunately, I know him personally, and he knew very well that I would not make any such statement. I happened to meet him soon after the patient was here, and learned from him the particulars I have just communicated to you. I told him I did not think it was at all likely that any one connected with the Clinic had said anything of the kind. I told him that I had explained to the gentlemen who were present at the Clinic, the very serious nature of the case, the difficulty of arresting the disease, and the very unsatisfactory position in which we are in regard to the treatment of progressive muscular atrophy; and that it was consequently most unlikely that any one who had seen the patient at the Clinic could have made any such statement. I promised him that when the patient came back to the Clinic, I would try and clear the matter up.

Well, you have heard the patient's statement. It appears, as I had supposed, that nothing was said to her which could have formed a basis for such a statement. That is so far satisfactory. The case is an instructive one, for it shows how easily misunderstandings may arise, and how many misunderstandings based on the mere statements of patients may be prevented. Had the doctor not known me personally, he might perhaps have concluded that I had given the patient to understand that I considered that she had been improperly treated. Like a sensible man, he came to me and told me what he had heard, and asked me to inquire into the matter. Now that is the only satisfactory way of clearing up such misunderstandings. If you attach importance to all the statements which patients make, you will often do your fellow practitioners a great wrong. One should be very careful as to what one says to patients. You cannot be too careful as to what you say to them as regards another man's treatment. When a patient comes to consult you, it necessarily every now and again happens, that you take a different view of the case from the previous medical attendant, and that you consequently propose a different plan of treatment. That must necessarily every now and again occur. A patient has a right to consult any man he likes; and when a patient consults you he has a right to know what you think about his case; but he has no right to ask you to criticise the opinions and treatment of his previous medical attendants. One should not allow oneself to be persuaded to criticise another man's opinion and treatment to the patient. When a patient feels dissatisfied with one medical man, he has of course a perfect right to call in another. If you are called in under such circumstances, it is your duty to do the best you can for the patient, and to tell him honestly what you think, and what you advise, irrespective of anything which he may have been previously told or advised. But there your duty ends. It is not your duty to tell him that you think the previous opinion and treatment were wrong, even if you should happen to think so. One must remember, that differences of opinion must necessarily arise in practice. Owing to the complicated nature of the problems with which the medical man has to deal, owing to the inherent difficulties associated with the diagnosis of disease, and owing to the different views which different men hold, and quite legitimately hold, as regards treatment, even when the diagnosis is clear, it necessarily follows that differences of opinion must frequently occur. These necessary and legitimate differences of opinion are not, however, the differences to which I am at present referring. I am alluding rather to those cases, in which the opinion of the previous medical attendant is clearly and obviously wrong, or in which his treatment has clearly and obviously been unsuitable. Now it is not your business to tell the patient that you think the opinion or treatment of your predecessor was wrong. In fact, quite the contrary. All of us make mistakes, and you should do to others as you would wish to be done by. All you

have to do is to take up the case where you find it, and to honestly and conscientiously advise the patient to the best of your ability. You should endeavour to smooth over, rather than to aggravate, any dissatisfaction that he may feel, and in some cases perhaps rightly feel, with his previous medical attendant. If the patient drives you into a corner and presses you to say whether the previous opinion and treatment were erroneous or not, you should not allow yourself to pass a harsh judgment upon your predecessor. It is enough to let the patient know that you do differ; to tell him honestly and clearly what you think and what you advise; you should refuse to enter into a discussion as to the supposed demerits of your predecessor; and whatever you may think, you should not make disparaging statements about a fellow practitioner to the patient. It may sometimes be necessary and advisable to discuss such questions amongst one's professional friends; but, in my opinion, it is rarely if ever advisable, and it is seldom, if ever, necessary, to discuss questions of this kind with one's patients. A man who runs down and backbites his neighbours, may perhaps get a little temporary advantage with individual patients, but it is a dearly bought advantage; such conduct is sure to recoil upon him disadvantageously in the long run.

This case shows how careful we should be in attaching importance to the mere statements of patients. Knowing how erroneous their conclusions and deductions often are, how they twist and distort statements—often quite unconsciously and in good faith—one should be careful in accepting what they say with regard to the statements and opinions of other medical men. In very many cases, it is well to ignore second hand statements and opinions, to treat them as if they had arisen, as they so often do, in the mere imagination of the patient. If the matter seems too serious to be treated in this way, the man who supposes himself aggrieved, should courteously ask for an explanation. A carefully and courteously worded note, or better still a personal interview, provided always that it is likely to be conducted with tact and courtesy by both parties, will clear up the great majority of misunderstandings of this kind. One should either ignore the secondhand statement of the patient altogether, and attach no importance to it, and not allow it to rankle in the mind; or endeavour to clear it up by going at once to the fountain-head, and courteously asking the person who is supposed to have made the statement for an explanation. If this course were more frequently followed, a good deal of ill-feeling and unpleasantness might, I believe, be prevented.

In a future number of the *Studies*, I will take an opportunity of referring to some of the other causes of dissatisfaction and misunderstanding between doctors; and consider, in particular, the duties of the consultant towards the general practitioner.

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